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Elytroderma Disease of Ponderosa Pine in the Pacific Northwest

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Summary

The needle cast of *Pinus ponderosa* caused by *Elytroderma deformans* is a native disease that intermittently causes severe local damage. The fungus is perennial within the host, where it spreads vegetatively from one twig or branch to another. Even in its vegetative stage the fungus appears unusually sensitive to climatic stresses.

Foliage on infected twigs dies early in its second year, and the twigs usually die within a few years. Growth rates are reduced in approximate proportion to extent of crown damage. Uncrowded saplings and poles with healthy leaders can recover eventually from fairly severe infection if they are not attacked by other parasites while weakened. In mature stands, crown damage from extensive killing of twigs and branches is permanent, moderately infected trees become and remain more susceptible to root disease and beetle attack, and heavily infected trees are often killed directly by defoliation.

Despite its alarming appearance in outbreak centers, the disease is just another of the many normal hazards to which forests are subject. It does not threaten to exterminate ponderosa pine as a commercial species even on local areas, and usual good management practices should go far towards reducing its future inroads.

Contents

Introduction

The disease caused by *Elytroderma deformans* (Weir) Darker[1] has been conspicuous during recent years in many ponderosa pine (*Pinus ponderosa* Laws.) stands from southern British Columbia to northeastern California and western Montana. Although less destructive than its alarming appearance often suggests, it has reduced growth rates on more than 100,000 acres in Oregon and Washington and has killed many trees on a few areas of severe infestation, sometimes more than 1,000 acres in extent. Heaviest damage has occurred in central and northeastern Oregon.

This paper is concerned with the disease only on ponderosa pine and principally with its behavior and effects east of the Cascade Range in Oregon and Washington. It is generally less damaging in other regions and to other hosts.

[1] *Originally described by Weir (1916) as* Hypoderma deformans. *It is commonly called needle blight or needle cast, but "Elytroderma disease" seems preferable since it also invades and damages twigs, branches, and young trunks.*

Occurrence and Persistence of Outbreaks

Weir's (1916[2]) observations in 1913-15 were evidently made during the waning stages of an extensive outbreak that had started many years before. Damage from this or possibly a later and less extensive outbreak continued for several years in parts of southern Idaho. In Oregon and Washington during the 1920's and 1930's, large brooms (presumably relics of the outbreak described by Weir) were fairly common in some localities, but no damage was observed even where Weir had recorded the disease as destructive.

The current outbreak first attracted attention in 1945 in central Oregon. During the next few years, the disease was found to be occasional to common in stands of all ages throughout the region. Evidence to be presented later indicates that a heavy wave of infection occurred in the late 1930's or very early 1940's. Some subsequent infection by spores has occurred from time to time.

Severe damage has been confined to relatively small areas within a rather narrow altitudinal zone centered at about 5,000 feet in central Oregon (Hunt and Childs 1957) and 3,500 feet in northern Washington. By 1950, nevertheless, salvage of more than 100 million board feet of sawtimber became necessary on the Ochoco National Forest alone, and infection of pole and sapling stands was causing serious concern. The disease is less conspicuous today (1967), but it is still common, killing or stunting trees on areas where it was causing damage 20 years ago.

Almost simultaneous appearance of heavy infection in localities hundreds of miles apart indicates regionwide occurrence of some combination of weather conditions especially favorable for infection, but weather summaries have provided no clue to the origin of the outbreak. Contrary to Weir's (1916) experience, recent investigators have usually found it difficult to germinate spores (Lightle 1954) and infect trees experimentally (Roth 1959, Waters 1957); consequently, little progress has been made in studies of epidemiology of the disease.[3]

Persistence of outbreaks, however, is easily explained. Elytroderma invades twigs and buds as well as needles, survives perennially in the former, and spreads vegetatively into needle primordia (Sikorowski and Roth 1962, Waters 1962, Weir 1916). Thus, once a damaging wave of infection has occurred, the fungus is not dependent on annually recurring periods of weather favorable for spore germination and host invasion in order to continue causing damage for many years.

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[2] *Names and dates in parentheses refer to Literature Cited, p. 38.*

[3] *Gordon and Laurent report successful inoculations of needles not more than 3 weeks old (personal communication). They suggest that infection waves may occur when ascocarp development is retarded by unfavorable weather so that infected needles remain on the tree and produce ascospores in the spring.*

Description of the Disease

The most prominent feature of Elytroderma disease is the red-brown "flags" which appear in spring. On flagged twigs, many of the B needles[4] are reddened, usually throughout all except their basal one-fourth or one-half inch. Ordinarily, either all or none of the needles in a bundle are affected. Reddened bundles tend to be grouped rather than randomly intermingled with green ones, and to be most numerous toward the distal end of the twig segment. A needles are not reddened, although sometimes faded, by October. Older than B needles usually are not reddened (exceptions will be discussed later), but twigs flagged in the previous year have lost many of these older needles.

Several other diseases cause somewhat similar discoloration, but in spring and early summer an experienced observer can usually identify Elytroderma flags, even in tops of tall trees, by their color.[5] Reddened needles gradually fade and become partly obscured by new foliage; by late summer they are indistinguishable, at distances of more than a few feet, from old needles fading in the course of normal fall defoliation. Most of the faded needles drop from the tree during the winter.

In spring the reddened needles produce small translucent beads or fine tendrils of pycnidiospores, which persist until dissolved by rain or broken off. Dark lines appear on the outer (rounded) surfaces of the needles in May or June, become plainly visible as the season progresses, and by late summer develop into conspicuous black fruiting bodies (fig. 1, A and B) in which ascospores are produced.[6]

Elytroderma fruiting is sometimes sparse throughout much of the region. Distal parts of discolored needles then usually bear small, crowded, black fruiting bodies of another fungus possibly competitive with Elytroderma or even parasitic upon it.

Lesions characteristic of the disease (Lightle 1954) occur in the inner bark of infected branches and are plainly visible in branch segments from 2 to 10 or 12 years old (fig. 1, C, E, F, and G). Even in old branches that have just flagged for the first time, lesions are often abundant throughout all except the youngest segments and, at least in saplings, in adjacent parts of the trunk and in bases of unflagged branches in neighboring whorls. Slender stems invaded by Elytroderma soon become noticeably deformed.

Twigs once flagged usually flag again each spring and soon curve upward (fig. 2A). Vigorous infected branches on thrifty trees often develop into dense, irregular or usually roughly globose witches-brooms (fig. 2, B to D) which survive and

[4] A *needles are those produced during the current calendar year;* B *needles, during the preceding year.*

[5] *Winter-injured foliage, for example, is often mistaken for Elytroderma flagging, but is a paler red. Other differential characteristics of winter injury are its usually greater abundance in tops than in bottoms of crowns, usual restriction to the distal half of the needles, gradual instead of abrupt transition between killed and living tissues, and, of course, the absence of Elytroderma fruiting bodies. Unlike Elytroderma, winter injury and most other foliage diseases are seldom conspicuous in successive years.*

[6] *Ascospores cause new infections. Pycnidiospores are probably functionless. Darker (1932) describes and illustrates both kinds of spores and the fruiting bodies that produce them.*

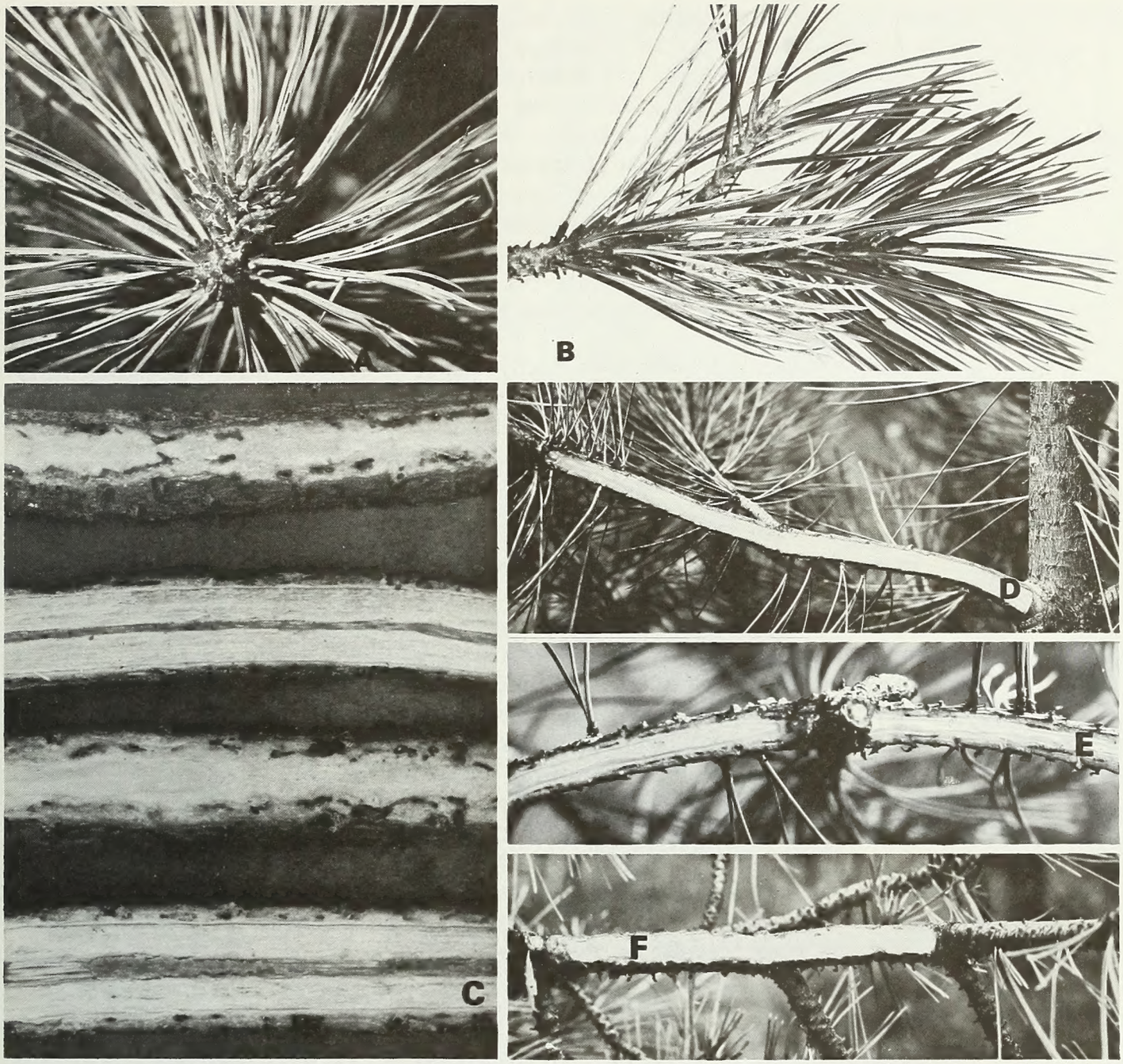

Figure 1. — A, Ascospore fruiting bodies of Elytroderma in late June (unusually conspicuous for so early in the year); B, ascospore fruiting bodies (dark lines) in late October (black dots are other fungi saprophytic on killed needles); C, tangential and radial sections of small branches showing necrotic lesions in inner bark; D, tangential section of healthy branch; E and F, tangential sections showing bark lesions in branch segments 3 to 4 and 9 to 10 years old, respectively; G, cross sections showing lesions in inner bark of young branches (section at upper right is from a healthy branch).

grow indefinitely. Flagged twigs on unbroomed branches gradually decline in vigor and seldom survive for more than a few years.

Variation from tree to tree in severity of infection undoubtedly is due partly to differences in susceptibility, but much of it obviously is associated with environmental differences. Infection is usually heaviest on pole and large-sapling understories, in the interiors of crowded groups of trees, and on lower branches and north to northwest sides of trees. Hunt and Childs (1957) found infection most abundant in stands of moderate density, at edges of meadows,[7] in wide draws, and on lower gentle slopes. These habitat preferences appear largely attributable to moisture requirements for spore germination, but some of them, together with other evidence, suggest that Elytroderma is relatively intolerant of high temperatures.

Small saplings probably were badly damaged in some localities during early years of the current outbreak but now are generally uninfected or only lightly infected. Trees that originated after about 1940 were not exposed to the main wave of infection, while many of the slightly older trees escaped it because of the small target they then exposed to infection. Despite the great numbers of ascospores since produced, infection is still not common on trees less than about 30 years old.[8]

In most localities, infection was never very damaging in any tree-size class, even though it was often conspicuous. Here the only signs of the disease have been the flags, still common on some trees, and in recent years, occasional to common brooming. Crowns of some thrifty young trees now consist of an outer layer of normal density formed by continued growth of uninfected branches, and a shorter, narrower, and denser inner part formed by broomed branches (fig. 2C).

Where infection was moderately heavy, most twigs in lower quarters or thirds of the crowns and many twigs higher in the crowns were flagged during early stages of the outbreak. Direct killing was rare here, but mortality increased from bark beetles[9] and apparently also from root diseases, often necessitating salvage logging. The unbroomed old flags are now dead, leaving crowns thin and ragged (fig. 2, D to G), often short, and spotted with newer flags.

Where infection was heaviest, entire lower crowns and many of the upper-crown twigs were soon killed. On some trees, more than nine-tenths of the twigs were either killed or flagged within a few years. Mortality became so common that mature stands were hastily logged except on small areas reserved for research. Many trees on reserved areas have since died and most survivors are in poor condition. In pole stands, survivors are partly compensating for destruction of lower crowns by adding new growth at the top, and often appear to be recovering.

In general, damage was considerably slower than at first expected, but it continued far longer and results were much the same even though the damage consisted less of rapid, direct killing than of progressive attrition of crowns and creation of high-risk trees.

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[7] *Probably because of the effects of these openings on temperature and humidity (Selleck and Schuppert 1957). Even in very dry periods, fog often forms at night over large "prairies" and then drifts into the neighboring forest (personal communication from R. W. Ellstrom).*

[8] *In 1949 and 1950, the disease was found on "trees of all sizes, from seedlings 6 inches high to mature trees. The youngest infected tree found . . . was determined to be 11 years old; only 1 such plant was found. Infected trees 12 years old were fairly common, but infection was more abundant in trees 14 years old and older" (Lightle 1954). In 1965 and 1967, two brief searches where the disease was common on large trees disclosed several old flags on trees 9 to 12 years old.*

[9] *According to personal communications from foresters in outbreak localities,* Dendroctonus brevicomis *populations increased in moderately infected stands more than in severely infected ones – presumably because saturated sapwood and sour smell of heavily defoliated trees make them unattractive to beetles.*

Figure 2. — A, Twig curvature and small brooms caused by Elytroderma; B, young broom; C, thrifty tree with numerous brooms in inner crown; D, mature tree with numerous small brooms; E, F, and G, crowns damaged by Elytroderma (flags in E appear white).

Studies and Plots

Early investigations of the current outbreak were limited to general observations since it was expected to subside within a year or so, as do most foliage disease outbreaks. Plot records consequently do not cover early stages of the outbreak. Studies were started in 1949 to determine the effects of different intensities of infection on mature stands. Damage plots were established in young-mature and old-growth stands where Elytroderma had caused little or no mortality but where severe damage was thought to be imminent. It soon became evident that data were also needed from smaller trees to help explain the behavior of the disease as well as to guide management of young stands.

This report is based principally on plots listed in table 1. Additional information was obtained from surveys, plots maintained only for brief periods, and tagged saplings used in fungicide tests. Data were also provided from five damage plots in mature stands in south-central Idaho; records indicated that effects of the disease there had been much the same as on the Oregon and Washington plots (see appendix).

Disease Behavior on Saplings

On the disease behavior plots, selected saplings more than 20 years old (and a few younger ones at Camas Creek) were examined annually in spring or early summer. Most of these trees had healthy leaders, were no more than lightly infected in the rest of the crown, and were no more than moderately crowded. Overstory density ranged from practically none at Camas Creek to light at John Day and Deer Creek.

Each branch with one or more flagged twigs was tagged, its position on the tree noted, its terminal (primary) twig recorded as flagged or healthy, and its flagged and total year-old and older lateral twigs tallied. Twigs were classed as "recovered" when they failed to flag after having formerly been flagged. Dead laterals were clipped to permit recognition of "recovered"[10] ones in subsequent examinations. General notes were taken on flag color, abundance and stages of development of Elytroderma fruiting bodies, and occurrence of fruiting bodies of other fungi on needles of flags. At Camas Creek, records of individual twigs were kept by means of branch diagrams. On the other two plots, regular data were supplemented by explanatory notes whenever changes in condition could be determined

[10] *Quotation marks are retained because subsequent behavior of many "recovered" twigs, branches, and trees indicated that they were still infected even though symptoms were not visible.*

TABLE 1. – LOCATIONS AND DESCRIPTIONS OF ELYTRODERMA STUDY PLOTS

Study and plot	Period of study	National Forest	Ranger District	Slope	Aspect	Elevation	Area	Number of trees	Average tree size[1]	
									D.b.h.	Height
						Feet	*Acres*		*Inches*	*Feet*
Disease behavior on saplings:										
1. John Day	1953-65	Malheur	Blue Mountain	Moderate	S.	4,900	([2])	70	--	7.6
2. Deer Creek	1954-65	Malheur	Bear Valley	Moderate	NW.	5,300	([2])	22	--	6.8
3. Camas Creek	1957-65	Umatilla	Ukiah	Moderate	SE.	4,700	([2])	67	--	7.5
Damage to poles:										
4. Dead Injun	1955-64	Malheur	Bear Valley	Gentle	SW.	5,200	10	281	7.5	--
5. Opal Flat	1955-65	Umatilla	Heppner	Level	--	4,400	7	224	9.5	--
6. Austin	1955-65	Malheur	Blue Mountain	Gentle	SW.	4,600	2	209	6.9	--
7. Chewiliken[3]	1956-65	--	--	Gentle	SW.	3,400	3	132	8.7	--
Damage to young-mature trees:										
8. John Day	1949-66	Malheur	Blue Mountain	Moderate	S.	4,900	5	110	17.5	--
9. Bannon	1956-65	Okanogan	Tonasket	Moderate	E.	3,500	4	115	13.5	--
Damage to old growth:										
10. Grays Prairie[4]	1949-65	Ochoco	Big Summit	Gentle	N.	5,200	28	335	27.2	--
11. Wildwood[4]	1949-65	Ochoco	Big Summit	Moderate	S.	5,000	6	177	26.7	--
12. Deer Creek	1951-65	Malheur	Bear Valley	Moderate	NW.	5,300	6	103	24.7	--
13. Belshaw	1951-66	Malheur	Long Creek	Moderate	SW.	5,200	5	159	23.8	--

[1] *At time of plot establishment.*

[2] *Indefinite.*

[3] *The Biles-Coleman Co. furnished assistance as well as study area.*

[4] *Strips 2 chains wide. All other plots were only about twice as long as wide.*

for individual twigs. Approximate numbers of uninfected branches and twigs upon them were computed from samples, as were tree height increments. A few additional data were obtained after the principal study on these plots had been terminated.

Numbers and conditions of trees, branches, and twigs (except branches and twigs in the few infected leader systems) are given in tables 2, 3, and 4 for each year of the study on the disease behavior plots. Here and elsewhere, uninfected classes include only units never recorded as flagged (some of these no doubt were invisibly infected). "Recovered" trees and branches are those once flagged but whose flagged twigs had all "recovered" or died. "Recovered" units are classed as infected.

Changes in amount of infection (except changes from additions to the basis in 1954, 1955, and 1957) were caused by appearance of new flags, death or "recovery" of flagged twigs, and death or relapse of "recovered" twigs:

Change from year before	Average annual change during study	
	Terminals	Laterals
	(Percent)	
New laterals[1] flagged on previously infected branches:		
On branches with infected terminals	–	78.9
On branches without infected terminals	--	11.5
Old twigs[1] flagged for first time:		
On branches previously infected	7.4	5.5
On branches not previously infected	.3	.2
Flagged twigs died	22.3	22.6
Flagged twigs "recovered"	6.9	5.0
"Recovered" twigs died	17.6	21.0
"Recovered" twigs relapsed	14.1	23.5
Uninfected twigs died on infected branches	8.0	7.7

[1] *New laterals are those just old enough to flag – that is, in their second growing season; old twigs are terminals and laterals in at least their third growing season.*

The much more frequent flagging of new laterals associated with infected terminals, as compared with those associated with uninfected terminals, substantiates Waters' (1958) and Sikorowski and Roth's (1962) conclusion that shoot primordia become infected vegetatively by growth of the fungus in the terminal bud. Other evidence indicates that the fungus also spreads vegetatively from twig to twig along branches, as suggested by Roth (1959), and even from branch to branch along trunks. Such spread requires consideration at some length.

Evidence of vegetative spread. — Of the 1,085 new flags recorded during the study, more than half occurred on branches previously flagged, even though such branches constituted only 11 percent of the branches on infected trees (7 percent of branches on total trees) and were often intermingled with branches, on the same or other trees, that never became flagged. On branches flagging for the first time, 59 percent of the flags appeared in groups of two or more per branch and

TABLE 2. – NUMBER AND CONDITION OF TREES ON DISEASE BEHAVIOR PLOTS, BY YEAR OF EXAMINATION

Condition of trees	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965
	Number of Trees												
Flagged — added to basis	36	12	17	0	21	0	0	0	0	0	0	0	0
Not flagged — added to basis	--	10	17	0	46	0	0	0	0	0	0	0	0
Flagged year before — still flagged	--	36	46	56	52	71	82	67	31	30	48	47	46
Flagged for first time	--	--	1	1	2	8	1	2	0	0	0	0	0
Relapsed	--	--	--	0	1	4	0	0	0	20	5	2	3
Flagged year before — "recovered"	--	0	2	8	5	5	1	16	38	1	2	6	3
"Recovered" trees died	--	--	--	0	0	0	0	0	2	0	0	0	0
Total flagged	36	48	64	57	76	83	83	69	31	50	53	49	49
Total "recovered"	0	0	2	10	14	15	16	32	68	49	46	50	50
Total uninfected	0	10	26	25	69	61	60	58	58	58	58	58	58

TABLE 3. – NUMBER AND CONDITION OF BRANCHES ON DISEASE BEHAVIOR PLOTS, BY YEAR OF EXAMINATION

Condition of branches	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965
	Number of branches												
Infected branches:													
On trees added to basis	79	22	31	0	60	0	0	0	0	0	0	0	0
Flagged year before —													
still flagged	--	75	104	153	142	207	226	162	60	50	111	111	110
Flagged for first time on:													
Tree already flagged	--	16	46	14	36	34	36	4	2	27	13	10	13
"Recovered" tree	--	--	--	0	0	6	0	0	0	13	5	3	3
Previously uninfected tree	--	--	1	1	2	8	3	3	0	0	0	0	0
Relapsed	--	--	0	0	1	5	2	1	1	40	11	3	2
Flagged year before — "recovered"	--	1	2	13	8	11	15	54	71	3	10	14	7
Flagged year before — died	--	3	7	16	18	23	19	51	39	10	9	15	10
"Recovered" branches died	--	--	0	0	2	1	3	4	15	22	5	1	8
Total flagged branches	79	113	182	168	241	260	267	170	63	130	140	127	128
Total "recovered" branches	--	1	3	16	21	26	36	85	140	81	75	85	82
Uninfected branches:[1]													
On flagged trees	425	636	843	807	1,348	1,609	1,658	1,555	811	1,231	1,300	1,264	1,278
On "recovered" trees	--	--	27	146	212	232	254	555	1,403	1,078	1,130	1,238	1,317
On uninfected trees	--	180	370	376	1,647	1,503	1,568	1,575	1,640	1,727	1,796	1,872	1,941

[1] *Estimated from samples.*

TABLE 4. – NUMBER AND CONDITION OF TWIGS ON DISEASE BEHAVIOR PLOTS, BY YEAR OF EXAMINATION

Condition of twigs	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964	1965
	Number of twigs												
Infected twigs:													
On trees added to basis	136	62	61	0	168	0	0	0	0	0	0	0	0
Flagged year before--													
still flagged	--	130	231	314	311	519	509	353	115	109	248	252	257
Flagged for first time on:													
Branch already flagged	--	42	29	45	68	109	77	41	18	42	32	35	39
"Recovered" branch	--	--	0	0	0	4	3	1	0	24	3	4	2
Branch previously uninfected	--	34	82	22	61	65	56	8	4	63	27	23	22
Relapsed	--	--	--	0	2	7	3	0	3	71	12	5	2
Flagged year before—"recovered"	--	0	5	10	6	14	28	50	113	1	6	11	3
Flagged year before — died	--	6	32	79	64	77	167	245	175	30	55	59	59
"Recovered" twigs died	--	--	--	1	2	0	4	15	27	29	9	4	10
Total flagged twigs	136	268	403	381	610	704	648	403	140	309	322	319	322
Total "recovered" twigs	--	0	5	14	16	23	44	79	162	63	48	50	41
Uninfected twigs:[1]													
On flagged branches	204	249	402	386	594	653	664	467	203	446	470	421	411
On "recovered" branches	--	2	7	37	47	63	87	217	407	269	254	286	259
On uninfected branches:[2]													
On infected trees	2,052	3,210	4,330	4,741	9,396	11,103	11,535	12,725	13,370	13,965	14,704	15,130	15,705
On uninfected trees	--	909	1,841	1,870	9,920	9,065	9,460	9,499	9,904	10,445	10,868	11,320	11,747

[1] *1 year old or older (twigs in their first growing season are not flagged by Elytroderma).*

[2] *Estimated from samples.*

30 percent in groups of three or more, even though less than 1 percent of the previously uninfected branches became flagged in an average year. On branches previously flagged, new flags also tended to appear in groups, usually adjacent to older flags.

During the 8 to 12 years they were under observation, more than 22 percent of previously uninfected branches became flagged on trees bearing old flags and nearly 5 percent on "recovered" trees, but only 1.4 percent on previously uninfected trees. Like newly flagged twigs, branches flagged for the first time tended to occur near previous flagging.

On 24 of the 98 trees flagged before 1959, visible infection remained confined to one branch (on 11 of these trees, to one twig) during the remainder of the study. On other trees it often skipped some twigs or branches more or less permanently. Nevertheless, nonrandomness of flagging was far too consistent to be attributed entirely or even largely to differences in microenvironments and tree susceptibility. Two examples of close association of new flagging of twigs and branches with older flagging are shown in figure 3.

In A to D of figure 4, bark lesions caused by Elytroderma are shown[11] in trunks and branches above the highest flag. In D and F of this figure, 3-year-old needles on one branch and 2-year-old needles on three branches and a leader are flagged where all younger needles have remained green. On twigs flagged more than once it is very rare for even a single 2-year-old or older needle to show signs of Elytroderma infection. On new flags, however, flagging of 2-year-old needles, followed by typical Elytroderma fruiting, is common; such flagging clearly indicates that the needles have been invaded by growth of the fungus from the base toward the tip of the twig instead of from meristem into primordia in the bud.

Extent and importance of vegetative spread. — Rate of vegetative spread could be estimated only in general terms, partly because it seemed to be strongly affected by bark age and condition, and partly because of evident variations in length of period between invasion of foliated twig segments and appearance of the flags.[12] In most trees on the disease behavior plots, spread rate appeared to be only a few inches — perhaps 4 or 5 — per year in the lower and central parts of the crowns, but two or three times as fast in vigorous tops and branches.

Distal spread is more common, or at least more rapid and extensive, than proximal spread. Branch infections in tops of young trees often spread to leaders (for example, as in figure 4F), making trees worthless for timber production. Of the 10 saplings whose leaders became flagged during the study, nine had branches previously flagged within 3 feet of the leader.

Thrifty saplings infected only in the lower crowns grow faster than the infections advance upward, and sooner or later are probably freed of visible infection through suppression of lower branches and action of other factors adverse to the disease. Long-term effects of stem deformation and phloem injury on quality and volume increment are unknown.

In larger trees, vegetative spread from branch to branch is probably not common, since initial infection by spores occurs at a greater average distance from the trunks than it does in saplings, and since growth of the fungus along branches is probably slower. However, larger trees have more twigs per branch, and twig-

[11] *Lesions are shown in figure 4 only where they could be attributed with reasonable certainty to Elytroderma. They are often hard to identify in old or long-infected bark. Diagrams* E *and* F *were drawn from photographs in which some of the details were obscure.*

[12] *For example, a badly stunted tree at Deer Creek remained for at least 11 years with only the two lowest branches flagged. Then, in 1966, its leader (about 2½ feet from the basal flags) and nine of its 10 intervening branches flagged simultaneously.*

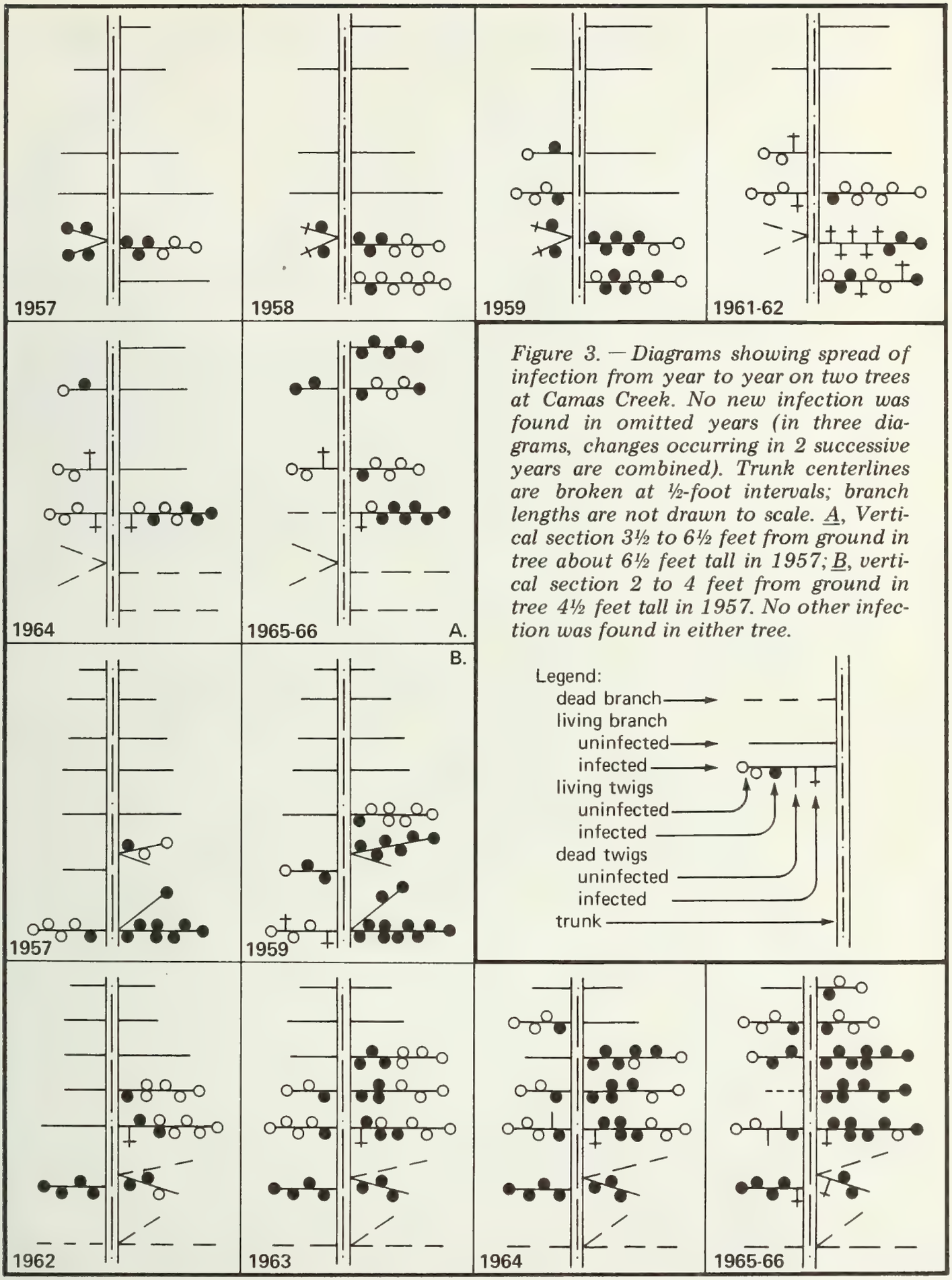


Figure 3. — Diagrams showing spread of infection from year to year on two trees at Camas Creek. No new infection was found in omitted years (in three diagrams, changes occurring in 2 successive years are combined). Trunk centerlines are broken at ½-foot intervals; branch lengths are not drawn to scale. A, Vertical section 3½ to 6½ feet from ground in tree about 6½ feet tall in 1957; B, vertical section 2 to 4 feet from ground in tree 4½ feet tall in 1957. No other infection was found in either tree.

killing is less rapidly compensated by new growth; consequently, a relatively small number of well-distributed initial infections may ultimately cause serious damage to large trees even if no branch-to-branch spread occurs.

Regardless of size of the trees involved, probably less than half of the total flags resulting from a wave of spore infection are visible at any one time. While intensity of infection seems to remain fairly constant as death of old flags more or less offsets the appearance of new ones, vegetative spread of infection, i.e., by growth of mycelium instead of by spores, continues to erode crowns whether or not additional spore infections occur.

Factors affecting persistence and spread of infection. — Twig death was the principal cause of decreases in numbers of flags. Of the 1,512 twigs flagged at one time or another in the study, 69 percent died while flagged and only 21 percent were still flagged in 1965. Death of about half of the killed flags was accompanied by death of the entire branch, often including twigs that had never flagged. Although suppression contributed substantially to flag mortality, mortality rate of infected twigs was more than twice that of uninfected twigs on infected branches.

"Recovery," especially of short spurs or suppressed twigs, was sometimes attributable to absence of B needles. Often, however, B needles were present but not visibly affected. Whatever its cause, "recovery" seems in most instances to have been merely temporary disappearance of symptoms rather than elimination of infection. Mortality rate of "recovered" twigs was nearly equal to that of flagged ones, and a majority of the survivors soon relapsed. Only 17 percent of the 247 "recovered" twigs were still alive and unflagged in 1965 (several of these, however, had been "recovered" for 4 or more years).

Numbers of dead, "recovered," and new flags all varied widely from year to year, but usually consistently with each other. Figure 5 shows that:

1. In 1956, flag mortality increased and new flagging decreased.
2. In 1957, mortality decreased at John Day (the basis at Deer Creek was small) and new flagging increased.
3. In the 1959-61 period, mortality and "recovery" rose to their highest points and new flagging dropped to its lowest points on all the plots.
4. In 1962, mortality and "recovery" again decreased and new flagging increased. (Relapse of "recovered" twigs reached its peak in this year.)

Corresponding changes occurred elsewhere in the region. In 1957, a large increase in number of flags, after a slight decrease in 1956, was noted by Roth (1959) on a study area about 125 miles from the nearest of the plots discussed here. In the 1959-61 period, the disease was less conspicuous than usual on trees of all sizes in most localities. In 1959, most of the discolored needles fell much earlier than usual. In 1960 and 1961, Elytroderma fruiting was generally sparse, fewer needles than usual were discolored on flagged twigs, and many of the discolored needles were affected for less than half of their length. Such consistent fluctuations in disease trends in widely separated localities must have been caused by regional rather than purely local fluctuations in weather.

Precipitation probably has little direct effect on vegetative spread of Elytroderma, but drought increases mortality rates of diseased twigs (Childs 1960). Heat treatments are used to control several plant pathogens that are killed by temperatures their hosts can tolerate, and some evidence suggests that unusually hot seasons retard Elytroderma spread and hasten death of infected twigs:

1. The disease's greater prevalence in shaded habitats and in the upper part of the altitudinal range of its host is correlated with temperature as well as

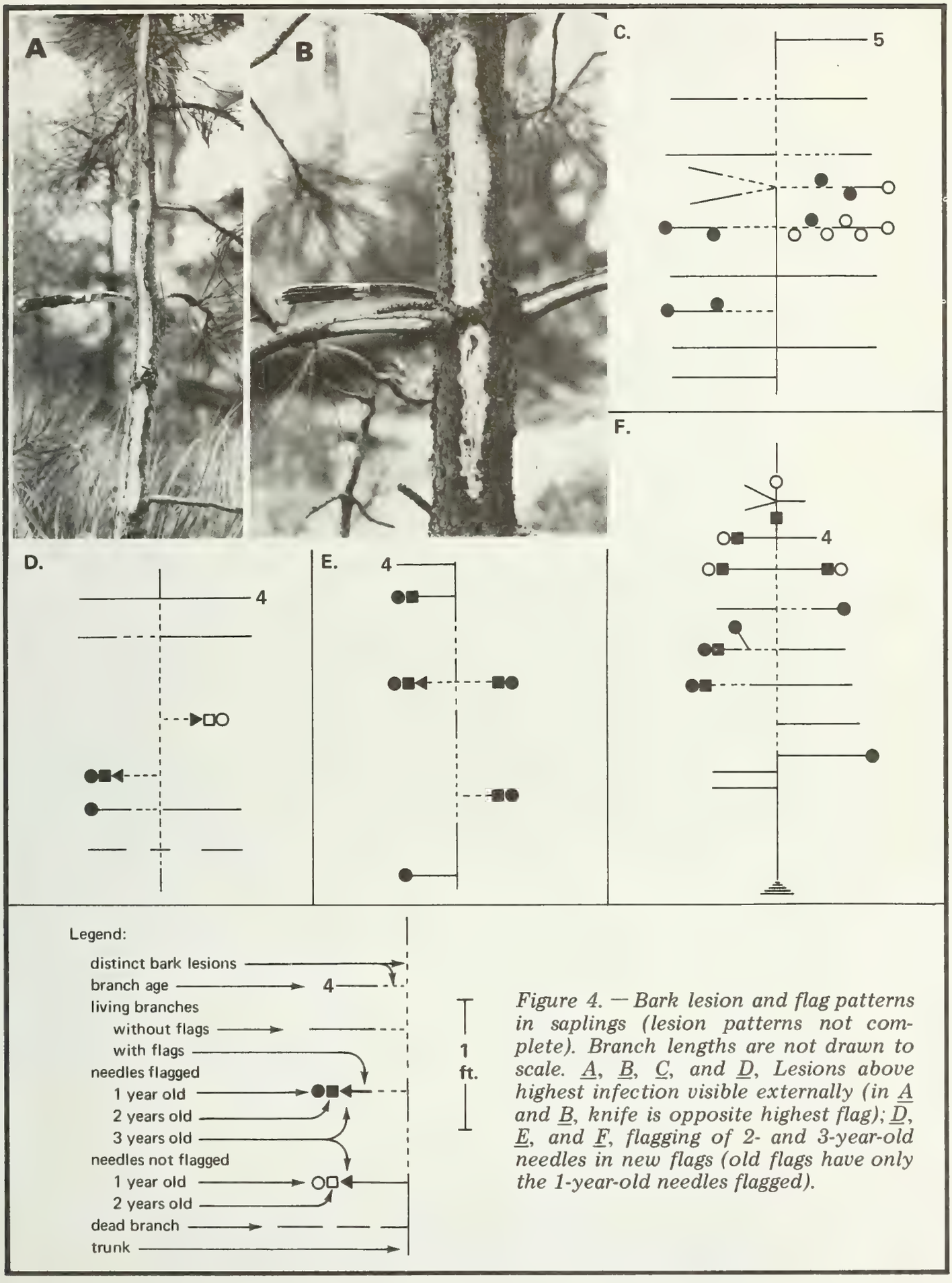


Figure 4. — Bark lesion and flag patterns in saplings (lesion patterns not complete). Branch lengths are not drawn to scale. A, B, C, and D, Lesions above highest infection visible externally (in A and B, knife is opposite highest flag); D, E, and F, flagging of 2- and 3-year-old needles in new flags (old flags have only the 1-year-old needles flagged).

moisture. In Roth's (1959) study, flags increased from 1955 to 1957 on saplings left in their original montane environment but disappeared almost completely from those moved to a lower and warmer location. "Outbreaks lasting only 1 or 2 years" have been reported (personal communications) from the Sierra Nevada in California but not from stands farther north.

2. The marked decrease in numbers of flags in the 1959-61 period followed unusually hot growing seasons in 1958 and 1960 and a relatively dry season in 1959. (Contrarily, however, the cool summer of 1955 was followed by decreases in numbers of new flags and increases in flag mortality, and recordbreaking heat in 1961 was followed by increases in new flagging and decreases in "recovery" and mortality.)
3. Decrease in number of flags during the 1959-61 period was greatest and subsequent increase was smallest on the hottest slope (John Day); at the same time the smallest decrease and greatest subsequent increase occurred on the coolest slope (Deer Creek). At John Day, decrease in number of flags was relatively greatest on southeast to southwest sides of crowns and least on northwest to northeast sides (fig. 6A). At Camas Creek it was greatest on east and southeast sides and least on northwest (fig. 6C). Changes in flag distribution on the northwest slope at Deer Creek (fig. 6B) are puzzling, but may have been caused by heat reflected from the opposite slope in this narrow valley.

Influences of weather may, of course, have been largely indirect — for example, through increasing the effectiveness of biological controls. Whatever the specific causes and their manner of action may have been, by 1961 the number of flags on the sapling plots had been reduced to a fifth of that in 1958 (table 4), and by 1965 had regained its former level only at Deer Creek (fig. 5D). After 1960, there was no increase in number of infected trees, and some of the "recovered" ones actually may have become free from infection. (Of the 68 classed as "recovered" in 1961, 38 were still unflagged in 1965.)

Though correlation of temperature with flagging is not proof of a cause-and-effect relationship, the weight of the evidence indicates that Elytroderma is more than ordinarily sensitive to climatic stress of one kind or another. Its harmlessness during the exceptionally hot, dry 1920's and 1930's therefore cannot be considered indicative of its potentialities under ordinary conditions.

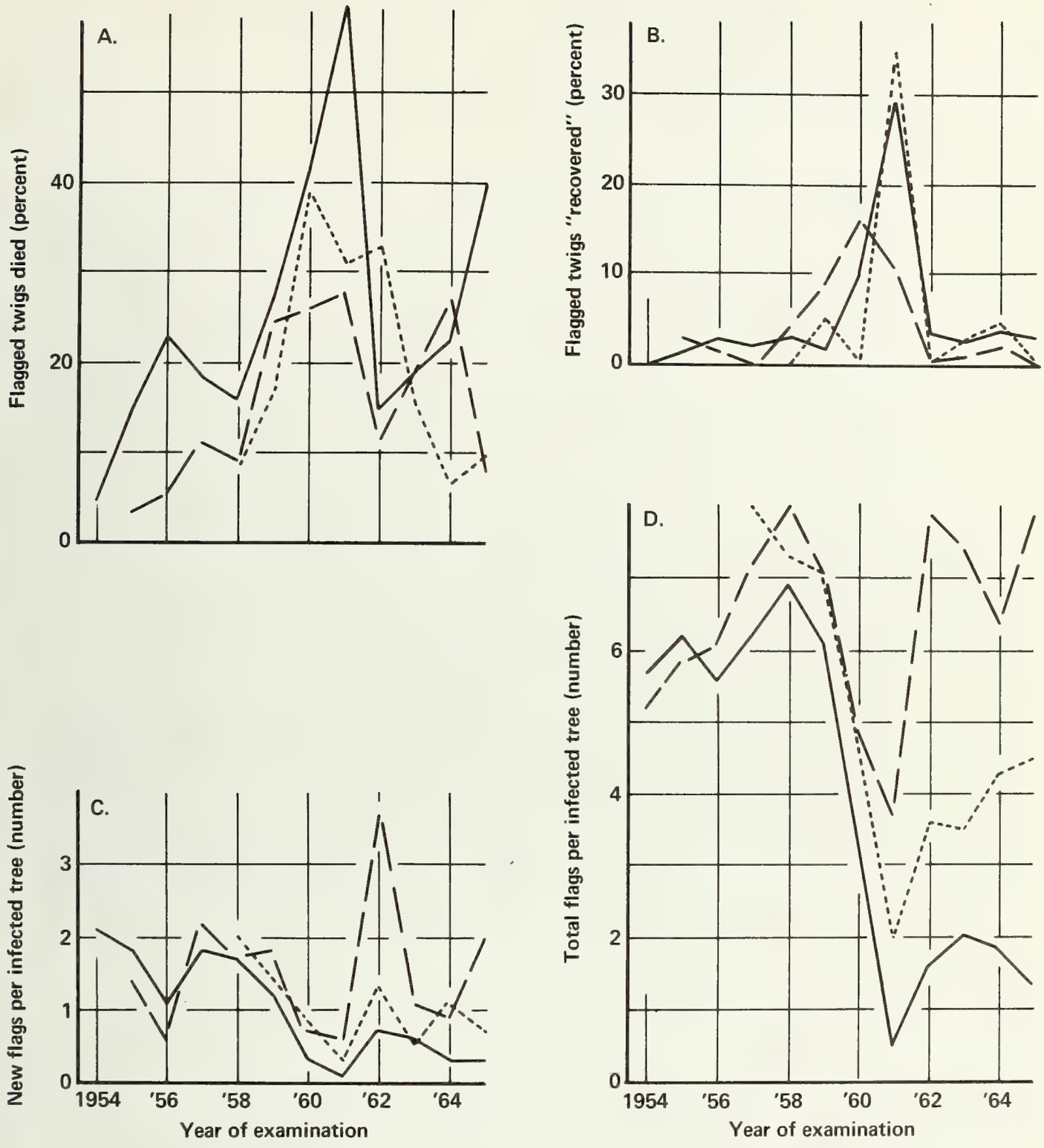
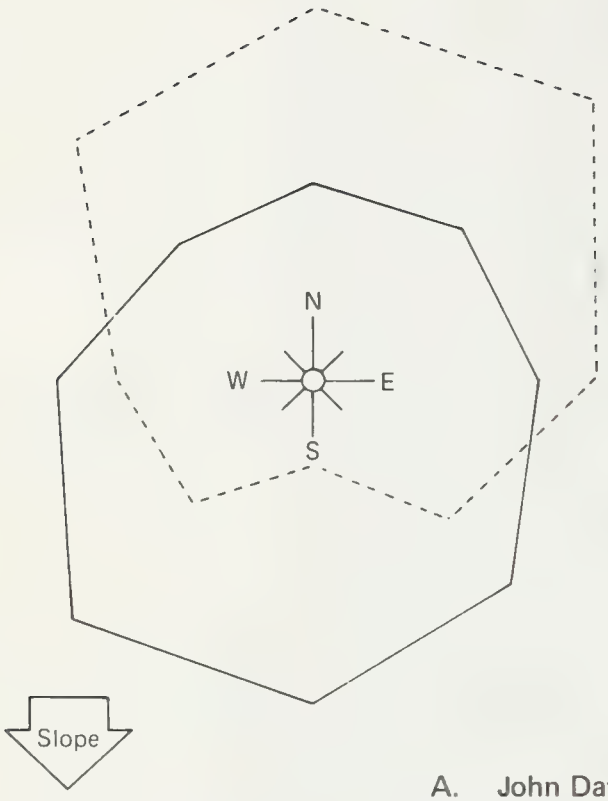
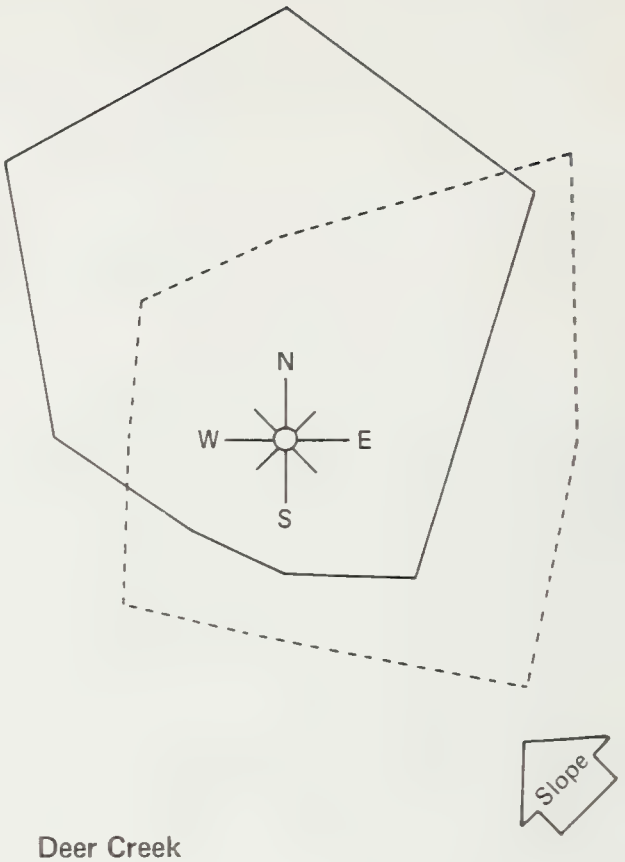


Figure 5. — Changes in flagging on sapling plots. Solid lines represent the John Day plot; broken lines, Deer Creek; dotted lines, Camas Creek.



A. John Day



B. Deer Creek

C. Camas Creek

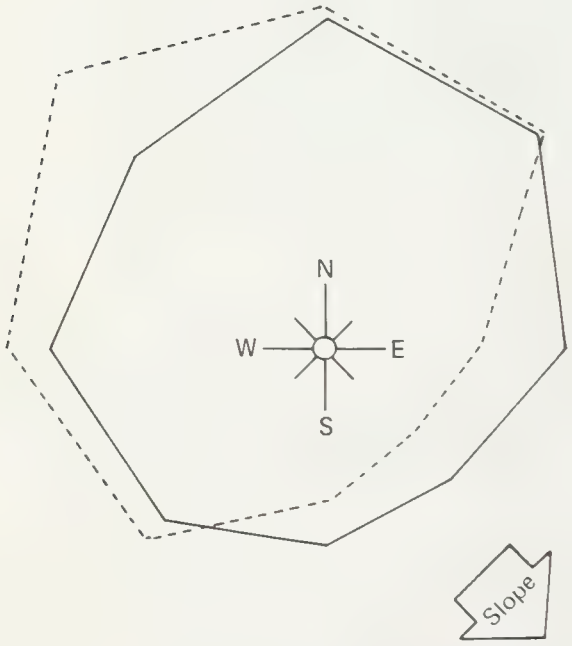
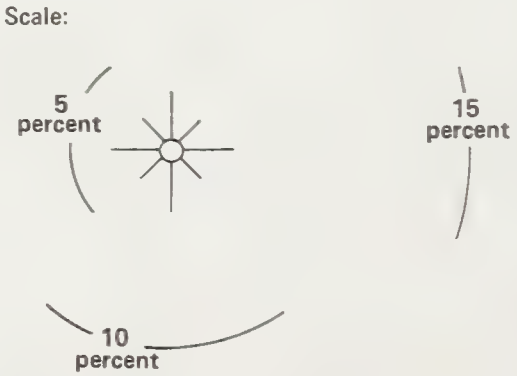


Figure 6. — Distribution of flags in 1958 (solid lines) and 1961 (dotted lines) on sapling crowns. Distances from diagram centers are proportional to percentages of total flags occurring on north, northeast, east, etc., sides of crowns (sums of distances along the eight radii equal 100 percent for each year). Arrows point downhill.



Damage to Older Stands

On the four pole plots (table 1), crop trees were marked with painted numbers and examined at 2- to 6-year intervals. Increment cores were taken from most of the survivors at Opal Flat and Chewiliken in spring of 1965.

On the two young-mature plots, all pines more than 6 inches d.b.h. at Bannon and 10 inches (including a few old-growth trees) at John Day were numbered and examined at irregular intervals (annually until 1952 at John Day). Increment cores were taken from most of the survivors at John Day in late 1955, and from all survivors at Bannon in the spring of 1965.

On the four old-growth plots, all pines more than 12 inches d.b.h. (including a few young-mature trees) were numbered and examined annually until 1952 and at irregular intervals thereafter. Increment cores were taken from most survivors except at Deer Creek in late 1955.

On all damage plots, percentages of living twigs flagged and (after 1952 or 1953) percentages of twigs dead were estimated for lower, middle, and upper thirds (by length) of each crown. D.b.h. was recorded in the first examination, and crown-condition class (Keen 1943) in the first (except at Chewiliken and Bannon) and last examinations. On the John Day and old-growth plots, estimates were also made of crown-condition classes as of about 5 years before plot establishment. Where good views were obtainable, trees on most of the plots were photographed in color at 4- to 10-year intervals.

Numbers of trees by flag-percentage classes at the first examination are shown in table 5 for each of the damage-study plots. Although lightly and heavily infected trees were intermingled to some extent, average intensity of infection differed considerably from one part of a plot to another. At Chewiliken and Bannon, infection was heaviest around grassy openings. On plots not complicated by openings or variations in stocking, infection was heaviest at the lowest end or corner:

Plot and elevation (feet)	Trees (Number)	Twigs flagged (average of first two or four examinations) (Percent)
John Day (1949-52)		
4,800-4,850	20	61
4,850-4,900	44	48
4,900-4,950	33	45
4,950-5,000	13	22
Wildwood (1949-52)		
4,800-4,850	23	10
4,850-4,900	12	5
4,900-4,950	24	1
4,950-5,000	25	1
5,000-5,150	93	1

Plot and elevation (feet)	Trees (Number)	Twigs flagged (average of first two or four examinations (Percent)
Belshaw (1951-52)		
5,100-5,150	5	81
5,150-5,200	61	53
5,200-5,250	54	42
5,250-5,300	39	32

Percentage of twigs flagged provides a rough index of infection intensity, since heavily infected trees will ordinarily bear many flags even after large parts of their crowns have been destroyed; but this percentage provides no measure of the extent to which twig killing has injured crowns. In an effort to obtain a better measure, crown damage percentages were computed from percentages of twigs flagged and percentages killed (dead twigs still present minus allowances for deaths from suppression). But this percentage, too, is only roughly proportional to total inroads of the disease. Where infection has long been present, many of the killed twigs and small branches have disintegrated, leaving no indication of time or cause of crown deterioration. (Computed percentages of crown damage sometimes decreased from one examination to another even when photographs showed that actual crown damage had increased.) Flagging and crown damage percentages are used interchangeably as convenient in the following discussion.

TABLE 5. — NUMBERS OF TREES BY INFECTION CLASSES ON DAMAGE-STUDY PLOTS

Plot	Percentages of twigs flagged at first examination									
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	91-100
	Number of trees									
Pole plots:										
Dead Injun	76	71	52	29	13	12	14	9	5	0
Opal Flat	130	46	25	8	9	3	3	0	0	0
Austin	160	28	11	2	2	4	0	2	0	0
Chewiliken	18	22	12	18	18	14	18	9	3	0
Young-mature plots:										
John Day	12	7	13	14	13	16	17	5	9	4
Bannon	25	10	11	15	11	8	12	9	7	7
Old-growth plots:										
Grays Prairie	202	60	31	11	10	7	10	2	1	1
Wildwood	171	2	3	0	0	0	0	0	0	1
Deer Creek	50	18	11	8	5	8	2	1	0	0
Belshaw	29	16	18	16	10	16	12	26	11	5

Average flagging and crown damage percentages at different times in the study are summarized in table 6. Inclusion of trees that subsequently died would raise some of these percentages; at Belshaw, for example, average crown damage in 1952 was 47 percent for trees that survived through 1965, and 52 percent for all trees. In general, the long-term effects of the disease on the more heavily infected plots were substantially greater than appear in this table. At Belshaw, Elytroderma had destroyed probably more than 60 percent of the crown of the average surviving tree by 1964; if this plot had not been under observation for the preceding 13 years, only a little more than half of this damage could have been charged with reasonable certainty to Elytroderma.

Although the average percentages in table 6 are fairly consistent with general observations and with growth and mortality effects of the disease, they sometimes fail to show changes that are known to have occurred. At Chewiliken, for example, where many new flags were observed in 1958 and some additional ones in 1964, the 1958 estimates showed an infection increase but in 1964 the new flagging had been more than offset by death of old flags, shedding of old killed twigs, and to some extent by growth of upper crowns.

Most new flagging apparently resulted from vegetative spread of the fungus in the vicinity of infections previously observed, but appearance in occasional years of new flags widely scattered in previously uninfected upper crowns indicated that new infections by spores also occurred from time to time.

Crown condition.— Keen's (1943) crown condition classes A to D were assigned numerical values from 4 (excellent) to 1 (poor), and average ratings were computed for each plot. Decreases in crown-condition ratings on pole plots were partly chargeable to suppression of lower crowns; even on these young trees, however, heavy infection had hastened and extended the dying of the lower crown, thinned much of the remaining crown and, by reducing growth rate, retarded the formation of new crown at the top to replace that lost at the bottom.

On young-mature and old-growth plots, rapid crown deterioration occurred only where infection was moderate or heavy:

Plot	Twigs flagged at first examination	Average crown-condition ratings of trees still alive at last examination	
		First examination	Last examination
	(Percent)		
John Day	0-40	2.9	2.7
John Day	41-90	2.7	2.1
Bannon	0-40	--	2.3
Bannon	41-98	--	1.5
Grays Prairie	0-40	2.5	2.8
Grays Prairie	41-98	2.6	2.6
Wildwood	0-40	2.4	2.3
Deer Creek	0-40	2.8	2.4
Deer Creek	41-80	2.8	2.0
Belshaw	0-40	2.3	1.9
Belshaw	41-90	2.2	1.4

Crowns at John Day and Belshaw also appeared to have deteriorated appreciably (about 0.3 on the numerical scale) during the 5 years immediately preceding plot establishment.

TABLE 6. – CHANGES IN FLAGGING AND APPARENT CROWN DAMAGE ON DAMAGE-STUDY PLOTS[1]

Plots and number of trees	Year[2]							
	1950	1952	1954	1956	1958	1960	1964	All exams
	Percentage of average crown							
Pole plots:								
Dead Injun (239):								
Flagged	--	--	24	--	24	--	25	24
Damaged	--	--	31	--	46	--	42	40
Opal Flat (217):								
Flagged	--	--	12	--	8	--	12	11
Damaged	--	--	16	--	22	--	20	19
Austin (199):								
Flagged	--	--	7	--	5	--	2	5
Damaged	--	--	13	--	29	--	25	23
Chewiliken (99):								
Flagged	--	--	--	29	44	45	30	37
Damaged	--	--	--	35	47	55	49	46
Young-mature plots:								
John Day (90):								
Flagged	43	42	26	28	18	--	14	28
Damaged	--	--	39	39	39	--	25	36
Bannon (87):								
Flagged	--	--	--	34	48	36	29	37
Damaged	--	--	--	38	52	45	44	45
Old-growth plots:								
Grays Prairie (322):								
Flagged	13	10	8	--	6	--	16	11
Damaged	--	20	14	--	16	--	25	19
Wildwood (158):								
Flagged	2	1	2	--	2	--	2	2
Damaged	--	7	10	--	11	--	10	9
Deer Creek (95):								
Flagged	--	22	21	21	21	--	11	19
Damaged	--	33	33	30	--	--	20	29
Belshaw (105):								
Flagged	--	37	29	24	17	--	23	26
Damaged	--	47	46	38	38	--	35	41

[1] *Includes only trees still alive at last examination. Excludes a few trees pruned, deformed, heavily infected by dwarf mistletoe, or with tops girdled or badly broken.*

[2] *Several percentages are combined from estimates made in 2 or occasionally 3 successive years, and a few are from single examinations made 1 year before or after the indicated date.*

Uncrowded saplings and poles with badly damaged crowns but good tops can recover fairly rapidly through upper-crown growth, but probably remain, for more than a decade, highly susceptible to the various parasites that prey on weakened trees. Larger trees, especially old-growth, do not repair damaged crowns rapidly. In sawtimber stands, even a single wave of Elytroderma infection contributes, proportionately to its severity, to more or less permanent deterioration of crowns and consequent reduction of growth and increase in vulnerability to bark beetles and other enemies.

Growth rate. — Growth data from Chewiliken increment cores were analyzed in greater detail than those from other plots. Table 7 shows that:

1. Correlations of d.b.h. with crown damage (column 3; variables B to E) were so small and lacking in statistical significance that they can be ignored.
2. Correlations of d.b.h. with 1955-59 and 1960-64 ring widths (column 3; variables M and N), although significant at the 5-percent level in the latter instance, were not great enough to invalidate comparisons between groups of trees differing moderately in average size.
3. Correlations of the four expressions of total crown damage (variables B to E) with 1955-59 and 1960-64 ring widths (columns 4 and 5) were all significant at the 1-percent level, but the maximum estimate (D) and the average of all estimates (E) each accounted for about one-fourth more of the variability in ring width than did either the 1956 or 1965 estimate alone.
4. Correlation of 1955-59 and 1960-64 ring widths with crown damage were improved only slightly by inclusion of upper-crown damage as another independent variable (variables M and N; columns 6 and 7).
5. Correlations of crown damage with ring width was also significant at the 1-percent level for the period 1950-54, but was not statistically significant for any of the preceding 5-year periods (column 6; variables G to L).

Elytroderma's depressing effect on diameter growth[13] is shown in column 8 of table 7, in figures 7 and 8, and in tables 8, 9, and 10. Although it is evident that the effect of the disease on growth became greater with passage of time, the data do not permit conclusions as to how much of the increase in effect is due to increase of infection and how much to cumulative impact of a constant intensity of infection over a period of several years.

Other complications also interfere with definite evaluations of the disease's effects on growth. For reasons previously given, flagging and crown damage percentages cannot be considered accurate measures of the inroads of the disease on crowns. In the small samples available, Elytroderma effects could not be clearly distinguished from effects of other factors. Root diseases (discussed further under "Mortality") apparently caused substantial reductions of growth rates in some instances — for example, in the five lightly to moderately infected trees represented by the broken line in figure 8B. Since Elytroderma is almost always most abundant in lower parts of crowns, light to moderate infection probably has more effect on mature trees than on poles, whose lower branches contribute little to tree nutrition.[14]

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[13] *Growth measurements were corrected for missing rings and for rare false rings by methods used by Keen (1937), McGinnies (1963), and others, and described in the appendix. The effect of the disease on growth was evident, however, before corrections were made.*

[14] *Pruning studies (Helmers 1946, Stein 1955) have shown that removal of the bottom quarter of live crown of young conifers has little or no effect on growth. However, diseased lower branches are perhaps net liabilities to the tree, rather than merely worthless.*

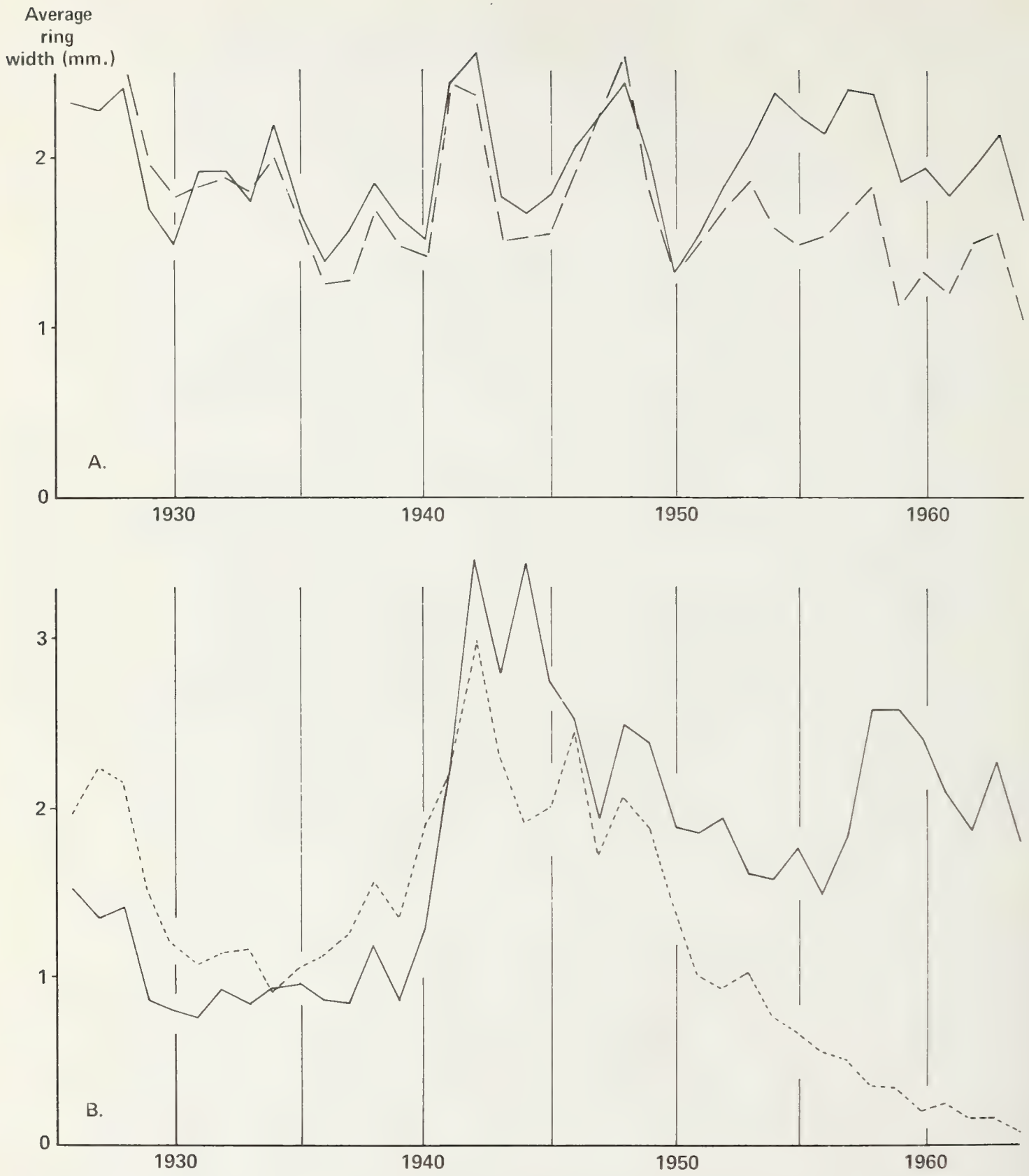

Figure 7. — Effect of Elytroderma infection on diameter growth in selected tree classes in pole stands. Solid lines represent light infection; broken line, moderate; dotted line, heavy. A, Opal Flat: 16 uninfected or lightly infected trees compared with eight moderately infected trees (the most heavily infected on this plot) of the same diameters (additional details in table 8); B, Chewiliken: the four most lightly infected trees (average crown damage 13 percent) compared with the nine most heavily infected surviving trees (average crown damage 76 percent).

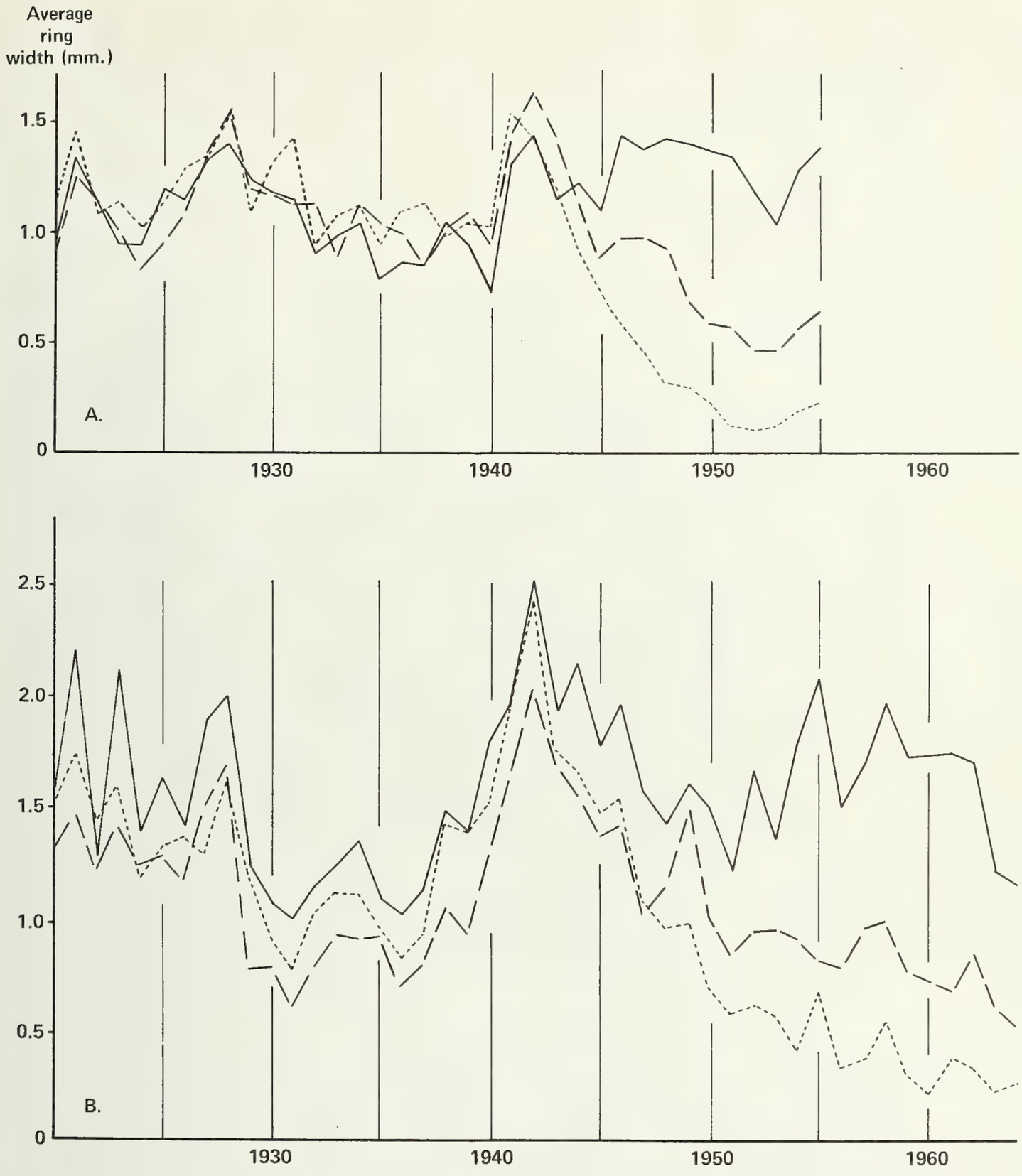

Figure 8. — Effect of Elytroderma infection on diameter growth in selected tree classes in young-mature stands. Solid lines represent light infection; broken lines, intermediate; dotted lines, heavy. A, John Day: trees with little or no dwarf mistletoe and 1921-30 average ring widths between 1.0 and 1.5 mm. (additional details in table 9); B, Bannon: four very lightly infected trees (average crown damage 6 percent) compared with five lightly to moderately infected (average damage 24 percent) and four heavily infected (average damage 71 percent) (all trees 15.1 inches d.b.h. or larger).

TABLE 7. — COEFFICIENTS BASED ON 94 CHEWILIKEN TREES STILL ALIVE AND AT LEAST 50 YEARS OLD IN 1965

Variable	Mean	Coefficient of					
		Simple correlation with variable--			Partial[1] correlation with variable--		Regression[1] on variable--
		A	M	N	E alone	E and F	E
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
A. D.b.h.	8.9 inches	1.0000	0.1833	0.2266[2]	--	--	--
B. 1956 estimate of total crown damage	34.7 percent	-.0245	-.6152[3]	-.6228[3]	--	--	--
C. 1965 estimate of total crown damage	51.4 percent	-.1294	-.5417[3]	-.6995[3]	--	--	--
D. Maximum estimate of total crown damage	59.8 percent	-.1079	-.6515[3]	-.7545[3]	--	--	--
E. Average estimate of total crown damage	47.6 percent	-.0773	-.6578[3]	-.7441[3]	1.0000	--	--
F. Average estimate of damage to upper crown	12.2 percent	--	--	--	.8594[3]	--	--
G. 1925-29 annual-ring width	10.5 mm.	--	--	--	.1584	--	0.034
H. 1930-34 annual-ring width	5.7 mm.	--	--	--	.1323	--	.020
I. 1935-39 annual-ring width	6.8 mm.	--	--	--	.0248	--	.004
J. 1940-44 annual-ring width	13.3 mm.	--	--	--	-.0603	--	-.018
K. 1945-49 annual-ring width	12.2 mm.	--	--	--	-.1973	--	-.052
L. 1950-54 annual-ring width	8.3 mm.	--	--	--	-.4368[3]	--	-.103
M. 1955-59 annual-ring width	5.5 mm.	.1833	1.0000	.8464[3]	-.6578[3]	-0.6602[3]	-.136
N. 1960-64 annual-ring width	4.0 mm.	.2266[2]	.8464[3]	1.0000	-.7441[3]	-.7492[3]	-.139

[1] *D.b.h. held constant.*

[2] *Significant at 5-percent level.*

[3] *Significant at 1-percent level.*

TABLE 8. — EFFECTS OF CROWN DAMAGE BY ELYTRODERMA ON DIAMETER GROWTH IN POLE STANDS

Plot and d.b.h. class (inches)	Average	Crown damage[1]			Tree basis	1925-29	1930-34	1935-39	1940-44	1945-49	1950-54	1955-59	1960-64	Average width of narrowest ring present[2]	Rings missing[2] in trees	
		Total Crown		Upper crown average												
		Range	Average												Rings	Trees
	Inches	*------Percent------*			*Number*	*-------Percent of growth of lightest infection class-------*								*Mm.*	*---Number---*	
Opal Flat																
(10.1-18.0)	13.4	0-14	5	< 1	16	100	100	100	100	100	100	100	100	1.01	0	0
	13.4	32-50	37	5	8	116	100	90	93	96	87	69	70	.74	0	0
Chewiliken[3]																
(6.1-9.0)	7.5	9-30	23	1	7	100	100	100	100	100	100	100	100	1.16	0	0
	7.6	31-50	41	6	21	111	98	101	99	74	71	54	55	.56	4	2
	8.0	51-66	58	16	16	108	104	92	94	76	64	37	24	.31	21	6
	7.1	72-80	76	39	7	114	108	108	85	74	50	28	12	.12	27	4
(9.1-12.0)	10.5	14-29	23	< 1	7	100	100	100	100	100	100	100	100	1.24	0	0
	10.6	31-49	39	4	8	118	120	110	111	107	89	66	62	.84	2	1
	10.3	51-69	60	19	14	125	116	114	119	97	62	36	28	.38	24	7
	10.1	74-80	77	44	2	105	117	62	89	48	26	10	6	.26	16	2

[1] *Average of all examinations.*

[2] *Since 1940.*

[3] *Trees at least 50 years old in 1965.*

TABLE 9. – EFFECTS OF FLAGGING BY ELYTRODERMA ON DIAMETER GROWTH IN YOUNG-MATURE STANDS

Plot	Average ring width 1921-30	Twigs flagged[1]		Tree basis	1921-25	1926-30	1931-35	1936-40	1941-45	1946-50	1951-55	1956-60	1961-65	Average width of narrowest ring present[2]	Rings missing[2] in trees	
		Class	Average												Rings	Trees
	Mm.	*----Percent---*		*Number*	*----------Percent of growth of lightest infection class----------*									*Mm.*	*---Number---*	
John Day[3]	> 1.5	< 30	18	11	100	100	100	100	100	100	100	--	--	1.24	0	0
		30-60	45	10	96	100	116	101	101	84	48	--	--	.55	2	1
		> 60	86	1	153	71	26	23	28	1	< 1	--	--	.08	9	1
	1.0-1.5	< 30	11	6	100	100	100	100	100	100	100	--	--	.86	0	0
		30-60	45	12	93	101	106	112	105	58	43	--	--	.42	18	4
		> 60	71	3	105	105	113	120	93	26	12	--	--	.07	0	0
	0.5-1.0	< 30	16	11	100	100	100	100	100	100	100	--	--	.45	0	0
		30-60	44	13	105	99	103	76	84	41	35	--	--	.15	21	9
		> 60	82	5	97	97	121	125	93	25	7	--	--	.13	31	5
	< 0.5	< 30	13	6	100	100	100	100	100	100	100	--	--	.36	0	0
		30-60	46	4	105	96	212	248	172	86	45	--	--	.17	4	2
		> 60	84	1	103	79	106	110	83	7	7	--	--	.04	4	1
Bannon[4]		< 30	16	9	--	100	100	100	100	100	100	100	100	.63	0	0
		30-60	41	11	--	91	103	112	103	113	89	71	71	.52	3	2
		> 60	71	4	--	93	101	108	102	83	48	35	27	.16	21	2

[1] *Average of all examinations up to year of growth measurement (Bannon figures are crown damage percentages).*

[2] *Since 1940.*

[3] *Exclusive of a few trees with moderate or heavy dwarf mistletoe infection.*

[4] *Trees more than 15 inches d.b.h. (not classified by ring width). Growth at Bannon is for periods 1925-29, 1930-34, etc.*

TABLE 10. – EFFECTS OF FLAGGING BY ELYTRODERMA ON DIAMETER GROWTH IN THE OLD-GROWTH STAND AT BELSHAW

Average ring width 1921-30 (mm.)	Twigs flagged[1]		Tree basis	1921-25	1926-30	1931-35	1936-40	1941-45	1946-50	1951-55	Average width of narrowest ring present[2]	Rings missing[2] in trees	
	Class	Average										Rings	Trees
	-----Percent----		*Number*	*--------Percent of growth of lightest infection class--------*							*Mm.*	*----Number----*	
>1.5	<30	20	8	100	100	100	100	100	100	100	0.67	4	1
	30-60	45	7	117	119	134	144	136	89	90	.64	12	2
	>60	70	6	89	102	94	109	87	30	20	.26	26	4
1.0-1.5	<30	15	10	100	100	100	100	100	100	100	.43	5	1
	30-60	49	10	91	103	105	132	144	78	42	.18	10	4
	>60	72	5	95	97	84	88	81	13	<1	.17	42	5
0.5-1.0	<30	13	33	100	100	100	100	100	100	100	.36	16	4
	30-60	51	12	94	94	121	107	93	59	35	.19	38	9
	>60	65	3	102	107	89	100	77	38	4	.27	26	3
<0.5	<30	13	15	100	100	100	100	100	100	100	.15	19	7
	30-60	49	13	111	113	100	104	115	62	43	.13	72	10
	>60	71	6	89	83	76	79	68	42	5	.07	47	6

[1] *Average of all examinations up to 1955.*

[2] *Since 1940.*

It may nevertheless be concluded, with due qualifications, that impact of Elytroderma on tree growth is proportional to extent of crown damage. This impact appears to be affected only slightly by age (compare tables 8, 9, and 10) or by vigor prior to infection (compare the different 1921-30 growth rate classes in tables 9 and 10).

Because of Elytroderma's persistence and vegetative spread, its impact on growth continues for several years, even in young trees. Most young trees probably recover eventually from fairly severe infection provided they escape other enemies, but growth-rate reductions as large and long-lasting as those shown in figure 7 and table 8 will considerably lengthen rotations.

Although volumes added by annual growth are less important in young-mature stands and of practically no importance in old growth, rates of growth in such stands are of interest because of their relationship to mortality risk. This relationship may sometimes be merely associative — that is, reduced growth may be only a symptom of a physiological weakening that predisposes the tree to disease or insect attack — but it may sometimes be causative. For example, resistance to certain root diseases probably depends in part on the rapidity with which the tree can form new roots or effective callus barriers on old roots. Whatever the relationship may be, slowly growing trees are notoriously poor mortality risks.

Mortality. — Field determinations of causes of deaths on the damage plots (table 11) are only roughly indicative of Elytroderma's mortality effects, since the disease was undoubtedly involved in some of the deaths charged to other causes — and, conversely, since some deaths charged to Elytroderma probably resulted from combinations of factors no one of which would have been fatal, or at least so quickly fatal.

Root disease effects, especially, could not be separated from those of Elytroderma. General observations during this and other studies suggest that root diseases most readily attack weakened trees and then often spread vegetatively to more vigorous neighbors. If this is true, Elytroderma may have indirectly caused death of some trees that it had infected only lightly. Well-known and fairly obvious root rotters such as *Armillaria mellea* and *Fomes annosus* appeared no more damaging where Elytroderma was abundant than elsewhere. However, an obscure and previously unobserved disorder, probably a disease of small roots, caused much crown deterioration and killed several trees at Bannon, on part of the Chewiliken plot, and perhaps also at Dead Injun, John Day, and Belshaw.

Although there is no way of knowing what mortality would have occurred on the damage plots if Elytroderma had not been present, it is evident from table 12 that heavy Elytroderma infection was associated with high mortality rates.[15] Whether killing was direct or indirect is of little importance. Annual mortality rates averaged 0.25 percent or less on plots where infection was generally light (Opal Flat, Austin, Grays Prairie, and Wildwood), but more than 2 percent where infection was heavy (Chewiliken, Bannon, and Belshaw). On all plots, mortality rates were highest in trees of the heaviest infection and poorest crown-condition classes. Similar results were obtained from plots in Idaho (see Appendix).

Mortality was nearly as common in large size-classes as in small. At Chewiliken, average d.b.h. was 8.7 inches for surviving trees and 8.2 for dead ones (again exclusive of trees windthrown); at Bannon, 13.1 and 11.4 inches; and at Belshaw,

[15] *Trees knocked down or windthrown are not included as mortality in this table, since their death could in no way be attributed to Elytroderma (except possibly through the effect of the disease on stand density). Mortality from all other causes is included since Elytroderma was a possible contributing factor in most instances.*

TABLE 11. – MORTALITY ON DAMAGE PLOTS, BY CAUSE

Item	Dead Injun	Opal Flat	Austin	Che-wiliken	John Day	Bannon	Grays Prairie	Wild-wood	Deer Creek	Belshaw
	Number of trees									
Alive at start of study	281	224	209	132	110	115	335	177	103	159
Death attributed to:										
Elytroderma	7	0	4	21	12	19	2	1	0	35
Root diseases	13	0	0	3	2	3	0	2	5	2
Beetles	2	0	0	0	4	0	2	2	0	5
Canker	0	0	0	2	0	0	0	0	0	0
Fire	0	0	0	2	0	0	0	0	0	0
Cut	0	2	0	0	0	0	4	0	1	3
Knocked or blown down	9	2	0	0	2	0	4	11	0	5
Unknown	0	3	0	0	0	0	1	2	1	4
Total dead	31	7	4	28	20	22	13	18	7	54
Contributing cause of death:										
Elytroderma	12	0	0	4	3	0	1	0	0	1
Root diseases	2	0	0	2	1	4	0	1	0	10

TABLE 12. – MORTALITY[1] ON DAMAGE-STUDY PLOTS

Plot	Crown condition class[2]	Total trees in infection class[3]				Trees died during study in infection class[3]				All classes	Length of study
		0-30	31-60	61-80	81-100	0-30	31-60	61-80	81-100		
		Number				*Percentage*				*Percent*	*Years*
Dead Injun	Good	168	48	17	5	4	0	12	40		
Dead Injun	Fair	27	6	6	0	15	50	33	--	8	9
Dead Injun	Poor	4	0	0	0	50	--	--	--		
Opal Flat	All	201	20	3	0	1	5	33	--	2	10
Austin	All	199	8	2	0	0	25	100	--	2	10
Chewiliken	All	52	50	27	3	8	8	63	100	21	9
John Day	Good	18	28	11	2	0	4	18	0		
John Day	Fair	14	13	9	5	7	15	33	40	16	17
John Day	Poor	0	2	2	6	--	100	50	67		
Bannon	All	46	34	21	14	7	9	33	64	19	9
Grays Prairie	All	293	28	12	2	2	7	0	50	3	16
Wildwood	All	176	0	0	1	3	--	--	100	4	16
Deer Creek	All	79	21	3	0	9	0	0	--	7	14
Belshaw	Good	18	14	4	0	6	0	25	--		
Belshaw	Fair	37	21	18	4	19	14	33	100	31	15
Belshaw	Poor	8	7	16	12	38	71	56	92		

[1] *Exclusive of trees knocked down or windthrown.*

[2] *At first examination. "Good" is Keen's (1943) classes* A *and* B; *"fair," class* C; *and "poor," D.*

[3] *Percentages of twigs flagged at first examination.*

23.8 and 20.5 inches. In general, trees killed by Elytroderma had grown more slowly than survivors (appendix table 13), not only since attack by the disease but also during the 1920's and 1930's. It is reasonable to assume that a given percentage of crown damage by the disease is most injurious to trees whose crowns are already in poor condition or whose growth rate has been reduced by other adverse factors.

Heavily infected trees are sometimes surprisingly tenacious of life. For example, when the John Day damage plot was established, two very heavily infected trees appeared unlikely to survive for more than a year; one of these trees survived for 7 years, and the other was still alive and apparently in slightly improved condition after 17 years. On this same plot, a tree 15 inches in d.b.h. had only seven living twigs in 1951 but survived until 1956, presumably because it was root-grafted to a less badly damaged neighbor.

Even though some heavily infected trees survive for many years, infection intensities like those at Chewiliken, Bannon, and Belshaw cannot long be tolerated in valuable stands.

Control

Lightle (1955) tested several chemicals but found neither surface nor penetrating fungicides to be effective against Elytroderma. Tests of systemic (penetrating) fungicides[16] in two Oregon localities were also unsuccessful, regardless of method (basal spray or foliage spray) or season of application. Some treatments, especially with stove oil as the vehicle, caused severe defoliation and some mortality, but surviving trees had the same twigs flagged after recovery from defoliation as before treatment.

Since injury by Elytroderma results largely from vegetative persistence and spread of infections, the disease seems highly vulnerable to chemical control if an effective eradicant suitable for aerial application can be found. At present, little can be done to reduce damage except to put young stands in thrifty condition, and to salvage threatened mature trees before they die.

Efficient salvage is not just a matter of harvesting moribund trees. The usual practice in ponderosa pine is to log all trees in poor condition but only enough of those in fair condition to bring the cut up to 30 or 40 percent of total stand volume. In Elytroderma-damaged stands, even where average infection was no more than moderate, such cuts have invariably been followed by rapid deterioration of residual crowns, necessitating relogging within a year or so. Additional cuts in quick succession have sometimes been necessary, leaving areas practically clearcut or with residual stands composed almost entirely of species other than ponderosa.

[16] *Phytoactin (provided by P-L Biochemicals, Milwaukee, Wis.) at concentrations of 200 and 400 parts per million in water, and various formulations of Actidione (provided by Upjohn Company, Kalamazoo, Mich.) at concentrations of 50 and 100 parts per million in water and in stove oil.*

Deterioration of residuals is fairly common in seemingly healthy stands and very common where diseases are prevalent. Like "shock" from thinning in young stands (Staebler 1956), this deterioration is probably caused by sudden and drastic environmental change which cannot be tolerated by trees already weakened.

Salvage of occasional dead and dying trees probably causes little damage to the remaining stand, since disturbance will be slight and some opening of the stand will occur whether the trees are removed or not. Where Elytroderma has made substantial inroads on the crowns and repeated cuts at short intervals would increase logging costs, it appears advisable to (1) postpone salvage until heavy mortality is imminent, beetle populations are increasing dangerously, or the stand is reached in the course of normal operations; and then (2) take all ponderosa pines except those of very good vigor (Keen classes A or B+) with no more than about 5 percent of the twigs flagged. On one of the last large salvage operations in a stand heavily infected by Elytroderma, more than 80 percent of the volume was cut and the remaining stand has continued in excellent vigor.

Discussion

Weir (1916) states that "average size of the brooms is about 2 feet in diameter" and "on large and mature trees *H. deformans* very rarely occurs on any part of the tree except the needles of these brooms." Since brooms from the present outbreak, approximately 25 years after it started, still average less than 2 feet in diameter, and since unbroomed flags are still common in large trees, the outbreak described by Weir must have started about 1890 or earlier. Furthermore, for several years prior to his observations, there seems to have been little or no spread of infection in large trees, either vegetatively or by spores, even though most years from 1900 to 1915 were moister than normal (Keen 1937).

Weir's observations were made too late in the outbreak to permit determination of the extent to which Elytroderma was responsible for crown damage and mortality, undoubtedly still evident at that time in pole and mature stands. Although he describes distribution and impact of the disease only in general terms, his report suggests that, as in the present outbreak, infection was common in many stands but seriously damaging in only a few.

Except for infection on lodgepole pine in one locality and old brooms, Elytroderma was not recorded again in Oregon and Washington for 30 years.

In 1945, when the current outbreak was first noticed, damage was already obvious on several hundred acres. Increment core measurements later showed that growth rates of some heavily infected trees had been reduced by 1944 and possibly as early as 1942. The disease evidently was present for some time prior to 1945, but was overlooked probably because of wartime diversion of forestry efforts.

Growth-rate comparisons indicate that the outbreak started at about the same time, both in Washington and Oregon, in all stands where appreciable damage was later caused. Growth of trees in moderately infected stands sometimes showed no effects until several years after 1944, but the same was true of moderately infected trees in heavily infected stands. Time from spore infection to flagging can hardly be less than a year, and Evenden (1940) found no retardation of basal growth of mature ponderosa pine until 1 year after "considerable foliage was destroyed." Therefore, even if the outbreak started with a wave of spore infections that gave rise directly to immense numbers of flags, this wave could not have occurred later than spring of 1942.

Origin of the outbreak in an initial wave of such magnitude is questionable. Elytroderma is poorly adapted for long-distance spread of infection in quantity, and local sources of infection were rare almost everywhere during the years just before the outbreak (although infection of lodgepole pine, where effects of this disease are less conspicuous, may have been more common than records indicate). It seems more likely, then, that great numbers of flags resulted from vegetative spread of the fungus from relatively moderate numbers of spore infections, or perhaps that a preliminary, moderate wave of infection provided sources of spores for a somewhat later major wave.

The main wave of infection by spores probably occurred no earlier than 1938 because (1) normal precipitation in eastern Oregon in 1938 was preceded by several dry years, when periods favorable for Elytroderma infection were presumably even rarer than in average or wet years; and (2) flagging from moderate or heavy infection that occurred before 1938 would undoubtedly have attracted attention while foresters were still engaged in peacetime activities.

During the last 15 or 20 years, intensification of infection appears to have been principally by vegetative spread along branches and, at least for short distances in small trees, along trunks. Additional infections by spores were common in occasional years, causing scattered flagging at considerable distances from original outbreak centers and increasing the severity of damage at the original centers. However, despite great numbers of spores produced and generally near-average precipitation since the early 1940's, damage by subsequent spore infections has probably been less than damage from the wave that occurred about 1940.

So much is still unknown about this disease, and especially about conditions governing origin and intensification of outbreaks, that prophecy is risky. Nevertheless, the facts available from later stages of two outbreaks provide some basis for predicting future behavior of Elytroderma.

Probably then, the current outbreak will continue its present slow abatement until its only evidences are unusually high percentages of poor-crowned trees in occasional stands, and old brooms and a few scattered flags here and there. After an indefinite period at this low level, the disease will again become conspicuous, simultaneously and perhaps suddenly in localized centers of heavy infection and gradually and less severely elsewhere. Within a few years, mortality rates will increase in moderately to heavily infected mature stands and in heavily infected pole stands, but severe damage will be confined almost entirely to the original centers of the new outbreak. After a decade or more, the cycle will be completed as flagging again decreases slowly in abundance.

Because of Elytroderma's marked topographic preferences, many of the centers of the current outbreak presumably were also centers of previous outbreaks. Yet previous outbreaks in these centers did not prevent (although they no doubt retarded) the development of mature stands that contained good volumes

of ponderosa when the current outbreak started. Previous outbreaks may, of course, have been less severe than the current one — but the current one has been no more than moderately damaging to originally thrifty young stands.

The disease is obviously too important to be ignored, but its importance has sometimes been overrated because of the sudden appearance and alarming aspect of the current outbreak. According to recent estimates, it causes less than 3 percent of the total damage by disease to ponderosa pine in this region. *Elytroderma deformans* is a native fungus, an inhabitant of ponderosa pine forests through countless forest generations. The balance that has developed between fungus and host is less stable than is usual with native fungi; but even where most favored by environmental conditions, the fungus does not threaten to eliminate its host as a commercial species. Like other normal hazards such as fire and beetles, the Elytroderma disease merits the forester's respectful attention but is no cause for dismay.

Recommendations

Young stands. — Since frequency and extent of future outbreaks cannot now be predicted, forest managers must rely on personal judgment in deciding how much weight to give the disease in management plans. Where infection during the current outbreak has been light, the disease is unlikely to be seriously damaging in the future. At the other extreme, stagnated stands on and near former outbreak centers are likely to be damaged by another outbreak before they mature. In such situations, Elytroderma disease may be one of the most important factors to be considered.

Thinning and pruning are too expensive by present methods to be justifiable solely for control of Elytroderma, but their probably mitigating effect on the disease is one of the benefits to be balanced against their costs in economic analyses of stand improvement. Greater investment risk where disease hazard is high must, of course, also be taken into account.

To minimize damage by infection now present or likely to occur before the stand matures:

1. Maintain good spacing. Infection is heaviest on crowded trees and most injurious to slowly growing ones. Large openings in the stand apparently make the environment more favorable for spread of the disease by spores.
2. Select no crop trees flagged within 3 feet and preferably none flagged within 6 feet of the leader. Infections near the leader often spread to it, and small trees with infected leaders seldom develop into anything better than "coniferous brush."
3. Select uninfected or only lightly infected crop trees, even at a considerable sacrifice of present tree size and quality. Flags visible during early stages of outbreaks are only a small fraction of those that will eventually result from

vegetative spread of infections already established. Infection in trunks of small trees deforms them at least temporarily, and perhaps permanently.

4. Prune where returns will equal investment plus carrying costs. The great majority of spore infections occur in lower crowns, where they produce quantities of spores for further spread, and from which they may invade and injure trunks and spread vegetatively for undetermined distances into higher branches.
5. Do not be bluffed by the disease. Where good pine has once grown, good pine can grow again.

Mature stands. — Future outbreaks will undoubtedly cause some damage even in vigorous young-mature stands and more severe and rapid damage in decadent over-mature stands. Since most pine stands are now fairly accessible, salvage difficulties are managerial and silvicultural rather than physical. The problem is to save threatened values without harm to the remaining stand and with minimum disruption of normal operations.

When the disease becomes conspicuous in mature stands:

1. Do not take hasty action. Appreciable damage will not occur for at least a few years after the outbreak starts. New flags in vigorous crowns are striking in appearance — much more so than killed twigs and old faded flags in poor crowns — but are not indicative of greatly reduced growth and imminent mortality.
2. Evaluate the situation annually on the basis of examinations made in spring and early summer, when flags are easiest to see and identify. Assume that:
   a. Flagged twigs will die within a few years.
   b. Additional flagging will occur — probably more than twice as much, ultimately and for the stand as a whole, as is visible during early outbreak stages.
   c. Trees with half of their twigs flagged or killed are likely to be attacked by bark beetles or root diseases, and trees with more than three-fourths of their twigs flagged or killed are likely to die as a direct result of defoliation.
   d. Trees with originally poor crowns will be damaged more rapidly and severely than equally infected trees with originally good crowns.
3. Revise logging plans to give higher priority to stands where appreciable damage is to be expected within the next few years, and to provide for immediate logging wherever mortality rates or beetle populations start to increase.
4. When logging in lightly infected stands, remove the occasional tree that may be found with more than a third of its twigs flagged or killed and discriminate as much as is practicable against the most heavily infected of the other trees.
5. When logging in moderately or heavily infected stands, either cut drastically, leaving only trees with very good (Keen class A or B+) crowns and no more than a few scattered flags, or plan to relog within a year or so if necessary. Partial cuts in diseased stands are usually followed by rapid crown deterioration and high mortality of residuals.

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Appendix

Estimating flagging and twig killing. — Although Elytroderma flags have a fairly distinctive color, it was occasionally impossible to make satisfactory estimates because of exceptional abundance of other foliage diseases. A few trees were chronically discolored by other diseases, and some were so screened by other trees that little of the crown could be seen clearly. Nevertheless, by use of various safeguards, estimates were obtained that proved in most instances to be accurate enough for purposes of this study.

Examinations were made between late May and early July, before the flags faded. Since the disease is not equally distributed on all sides of a crown, trees were viewed from opposite sides whenever practicable. Flagging can be seen best when the sun is behind or to the side of the observer. When crowns were silhouetted against an overcast sky, making flags hard to see, examinations were postponed or crowns were viewed through 2 mailing tubes fastened parallel to each other at the proper interpupillary distance (to exclude side light). Field glasses cause too much eyestrain when used all day.

The following precautions were taken against personal bias:

1. Estimates of different observers were checked against each other whenever two or more observers were available.
2. Estimates were often checked against those of previous examinations. Color photos were not very helpful in checking estimates, since they do not show infection as well as the observer can see it.
3. Occasionally, estimates were repeated on the same tree after an interval of an hour or more.
4. To correct the observer's tendency to be unduly impressed by red foliage, percentages of unflagged as well as flagged twigs were often estimated.

Missing rings. — Increment core measurements were corrected for missing rings as follows:

1. Trees with less than 30-percent crown damage, and with no evident condition likely to affect growth seriously, were grouped by size classes (Chewiliken and Bannon) or 1921-30 ring-width classes (John Day and Belshaw). Ring patterns of most of these trees were very similar and, with the exception of the most slowly growing trees, well marked by "key" rings. The few trees with obviously dissimilar patterns were excluded, and the remaining trees were assumed to have no missing rings.

2. Average ring-width for each of the last 35 or 40 years was computed for each group. Examples are shown in figure 9. With minor exceptions at Chewiliken and Bannon, the standard ring pattern features most consistent between individual trees, as well as between groups, proved to be the peaks of growth in 1921, 1928, 1934, 1941 or 1942, and 1947, and the low points in 1924 or 1925, most of the 1930's, and 1945. Occasional trees were 1 year out of synchronization with the majority at one or two of these key points.
3. Trees whose ring pattern did not closely coincide with the standard for their group were individually graphed on transparencies, which were moved back and forth over the standard graph to determine the position of best coincidence. A high degree of coincidence with the key points was usually obtainable; otherwise, the smallest correction that gave reasonably good coincidence was assumed to be the correct one. Among the most slowly growing trees, some showed no identifiable key rings, and consequently could not be corrected, while most of the remainder had no identifiable rings except those of 1921-22 and 1941-42.

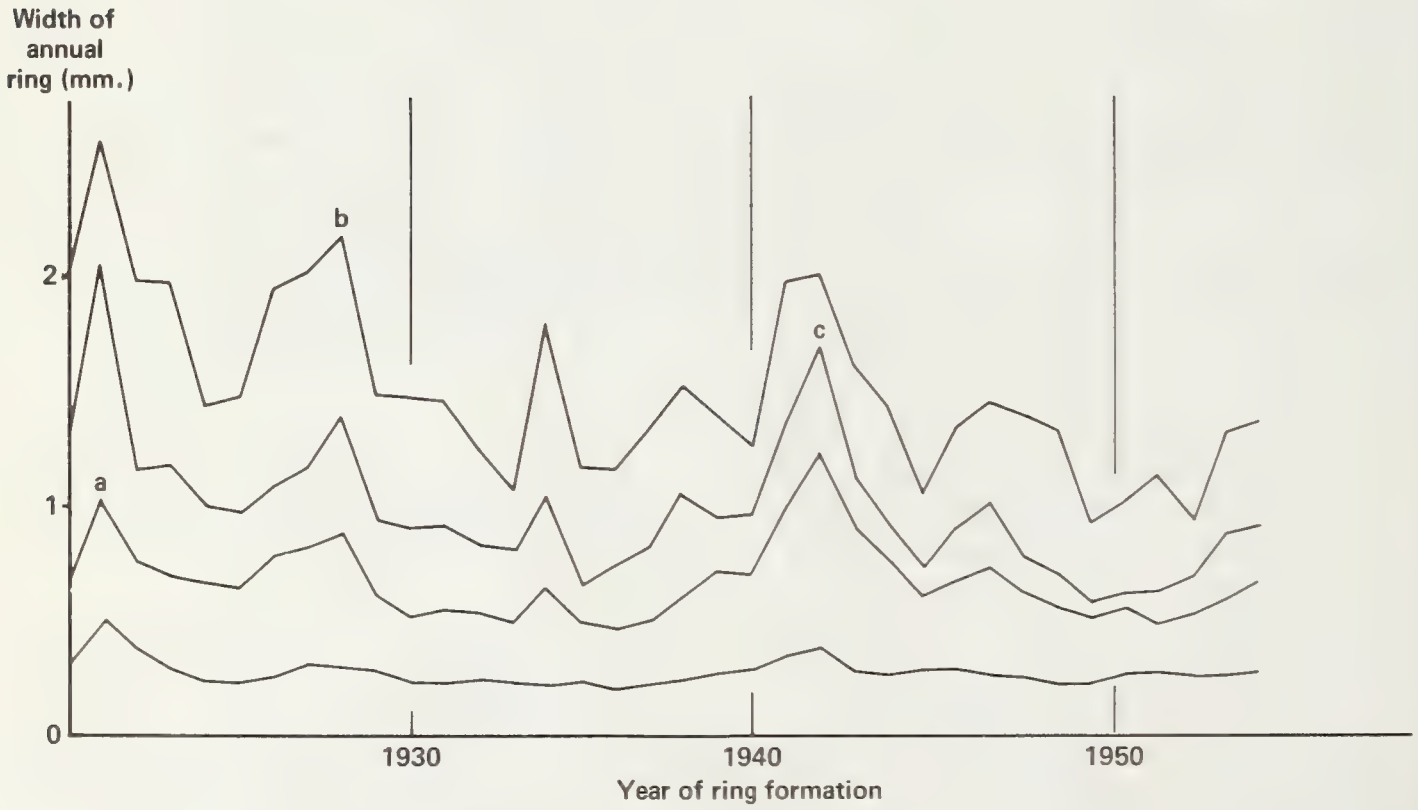

Figure 9. — Annual-ring patterns of Belshaw trees with no ring missing. Lowercase letters indicate three of the key points used in checking for missing rings. From top to bottom, lines represent seven trees with 1921-30 average ring-width more than 1.5 mm.; seven trees, rings 1.0 to 1.5 mm.; 26 trees, rings 0.5 to 1.0 mm.; and six trees, rings less than 0.5 mm.

Several of the indicated corrections seemed excessive even though the corrected patterns coincided closely with standard ones. Accordingly, cores were taken again in late 1959 from several moderately to heavily infected survivors at John Day and Belshaw. The 1955 and 1959 cores from one tree are graphed in figure 10. This tree apparently had had a good crown before Elytroderma attack, but by 1955 more than 80 percent of the crown was flagged or killed.

The 1955 core (broken line), as plotted in the graph, does not coincide with the standard at any key point except 1921 and 1928; if shifted 9 years to the left, however, it coincides not only with all key points (except, of course, the 1947 peak) but also with most of the minor irregularities in the standard pattern. The 1959 core (dotted line) by itself might have been interpreted as indicating an individual abnormality in 1925-26, one missing ring in the late 1930's, and one false ring but no missing rings after 1942; if shifted 4 years to the left, however, it coincides almost perfectly with the broken line. The two cores together indicate beyond reasonable doubt that, on the side of the trunk where the cores were taken, only four rings were formed from 1943 to 1955 and none from 1956 to 1959.

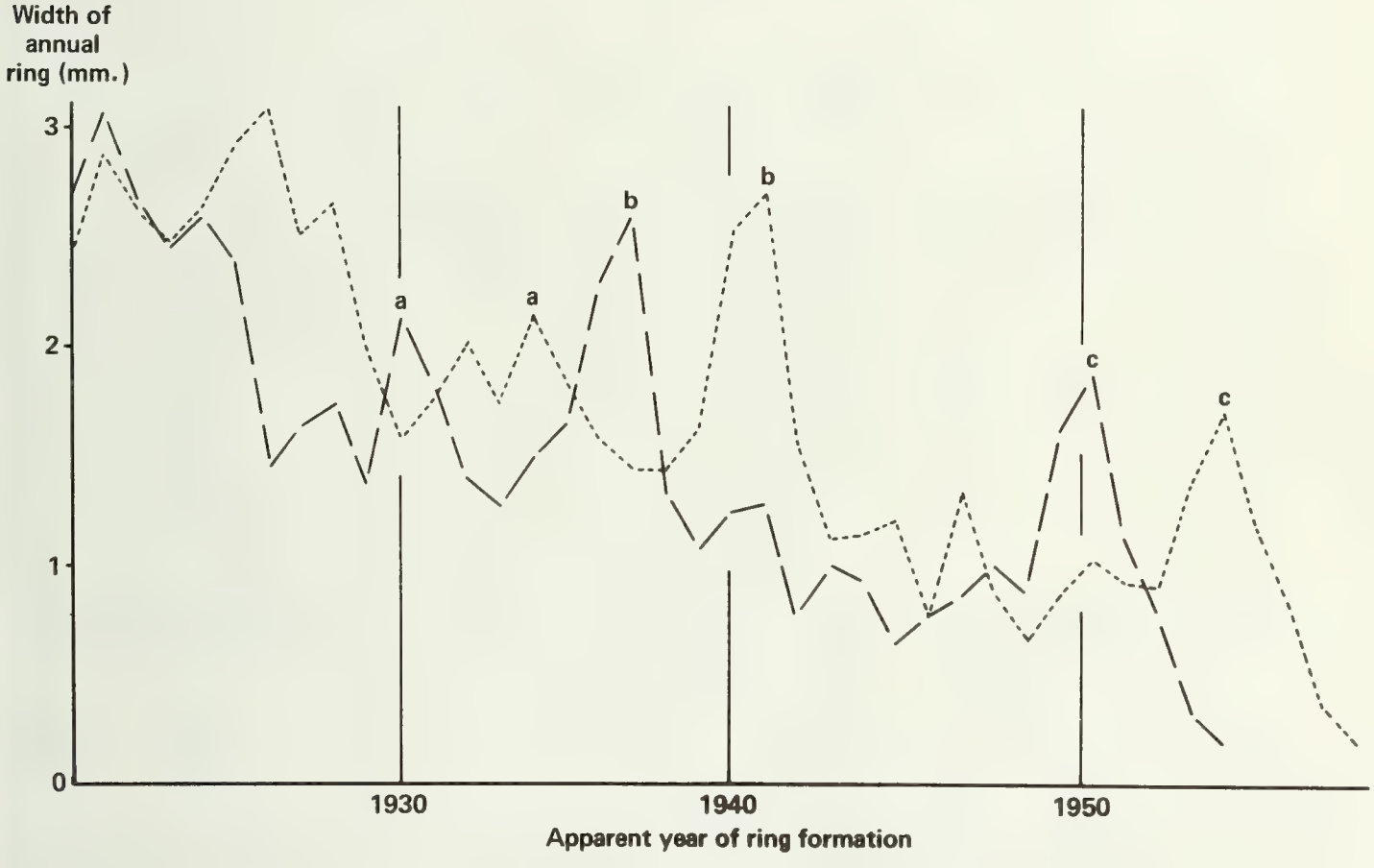

Figure 10. — Uncorrected ring patterns from heavily infected tree at Belshaw. Broken line represents increment core taken in 1955; dotted line, core taken from same tree in 1959. Lowercase letters indicate same key points as in figure 9.

As might be expected, several of the cores taken in 1959 had fewer rings missing than did the core taken from the opposite side of the same tree in 1955. When taken at a distance of only 8 inches from the 1955 core, all but two of the 1959 cores had at least as many rings missing as the 1955 core:

Plot	Trees	Average number of rings missing per tree	
		1955 core	1959 core
	(Number)		
Belshaw	15	0	0.2
	2	1	4.5
	3	2	3.7
	2	3	5.5
	3	4	5.2
	1	5	7.0
	3	6	9.3
	3	7	10.7
	2	8	10.0
	1	9	13.0
	2	10	12.0
	2	11	12.0
John Day	10	0	.2
	3	1	2.3
	4	2	2.8
	1	3	4.0
	1	4	5.0
	2	5	6.5
	1	6	9.0
	1	7	9.0
	1	9	13.0

Some tree characteristics associated with missing rings are shown in table 13. Evenden (1940) reports two instances of trees that made no basal growth for at least 11 years after heavy defoliation by the pine butterfly.

Disease effects on Idaho plots. — Probably the most severe damage by the disease outside of Oregon has occurred on the Boise and Payette National Forests in south-central Idaho. Plots to determine rate and extent of damage to mature stands were established on the Boise National Forest in 1951 (part of one plot in 1948) by the Intermountain Forest and Range Experiment Station. These plots, like the ones in Oregon and Washington, were not established until several years after the main wave of infection had occurred.

Average crown-condition ratings on these plots are given in table 14. Mortality by crown-condition and infection classes is summarized in table 15. Heavy mortality on plot 4 was largely caused by *Fomes annosus*, common here at time of plot establishment. On some of the other plots, Elytroderma infection appeared to have aggravated damage caused by an unidentified root disease.

TABLE 13.—RADIAL GROWTH AND MISSING RINGS IN TREES KILLED BY ELYTRODERMA AND IN TREES SURVIVING

Plot and group[1]	Class		Basis	Infection		Average annual-ring width				Missing rings[4]	
	Growth[2]	Infection[3]		Average	Range	1921-30	1931-40	1946-55		Average	Range
								Average	Range		
	Mm.	*Percent*	*Number*	*--Percent--*		*----------Mm.----------*				*----Number----*	
John Day:											
Killed trees	0.5-1.0	>60	2	88	87-88	0.68	0.87	0.01	0-0.03	9.5	9-10
Survivors			3	79	76-83	.79	1.00	.20	.05-0.46	4.0	2-6
Killed trees	>1.5	30-60	2	51	43-59	1.80	1.62	.86	.69-1.04	0	0
Survivors			8	43	33-59	2.03	1.90	1.36	.26-2.20	0.2	0-2
Belshaw:											
Killed trees	<0.5	30-60	3	54	47-57	.28	.28	.03	0-0.07	8.0	6-11
Survivors			10	48	34-59	.39	.27	.15	0-0.41	4.8	0-11
Killed trees	<0.5	>60	2	68	65-71	.24	.20	.06	0-0.12	8.5	4-13
Survivors			3	71	64-77	.33	.22	.02	0-0.05	8.7	6-11
Killed trees	0.5-1.0	30-60	1	59	59	.87	.92	.07	.07	7.0	7
Survivors			11	51	31-60	.70	.61	.28	0-0.82	2.8	0-11
Killed trees	0.5-1.0	>60	1	63	63	.88	.28	0	0	12.0	12
Survivors			2	66	62-69	.75	.65	.19	0-0.37	7.0	3-11
Killed trees	1.0-1.5	>60	2	67	65-69	1.33	.73	.03	0-0.06	8.5	7-10
Survivors			2	74	70-77	1.05	.86	.08	.04-0.12	7.5	7-8
Killed trees	>1.5	>60	2	73	64-82	1.68	1.12	.15	0-0.29	5.5	0-11
Survivors			3	70	61-81	1.77	1.18	.17	.02-0.36	5.0	2-9

[1] *Killed trees are those that died after 1955 with Elytroderma at least an important contributing factor if not the principal cause of death. Survivors are those still alive when study terminated.*

[2] *Average 1921-30 ring width.*

[3] *Percentage of twigs flagged (average of all examinations before 1956).*

[4] *Since 1940.*

TABLE 14. — AVERAGE CROWN-CONDITION RATINGS OF TREES STILL ALIVE IN 1966 ON IDAHO PLOTS

Plot number[1]	Twigs flagged (average of first three annual examinations)	Trees	Average crown-condition ratings[2]	
			1951 (or 1948) examination	1966 examination
	Percent	*Number*		
2	0-40	19	2.9	2.4
	41-67	4	2.2	2.0
3	0-40	20	2.8	2.7
	41-57	1	2.0	1.0
4	0-40	10	2.8	3.0
5	0-40	19	2.9	2.8
6	0-40	15	2.4	2.0
	41-55	3	2.0	1.0

[1] *Data for plot 1 were incomplete.*

[2] *4 = excellent; 3 = good; 2 = fair; 1 = poor.*

TABLE 15. – MORTALITY[1] BY INFECTION AND CROWN-CONDITION CLASSES ON IDAHO PLOTS

Plot number[2]	Crown-condition class[3]	Total trees in infection class[4]				Trees died during study in infection class[4]			
		0-30	31-60	61-80	81-100	0-30	31-60	61-80	81-100
		Number				*Percentage*			
2	Good	12	2	0	0	0	0	--	--
	Fair	3	5	2	0	0	20	50	--
	Poor	2	0	3	2	50	--	100	100
3	Good	12	2	0	0	0	0	--	--
	Fair	6	2	0	0	0	50	--	--
	Poor	0	3	3	0	--	100	100	--
4	Good	6	1	0	0	0	100	--	--
	Fair	3	6	0	0	0	83	--	--
	Poor	2	0	0	0	100	--	--	--
5	Good	12	1	0	0	8	0	--	--
	Fair	6	1	0	0	0	0	--	--
	Poor	0	1	0	0	--	100	--	--
6	Good	3	3	0	0	0	0	--	--
	Fair	5	10	1	0	0	30	100	--
	Poor	1	2	0	2	100	100	--	100

[1] *Exclusive of trees knocked down or windthrown.*

[2] *Data for plot 1 were incomplete.*

[3] *At first examination.*

[4] *Average percentage of twigs flagged in first three annual examinations.*

Childs, T. W.

1968. Elytroderma disease of ponderosa pine in the Pacific Northwest. U. S. D. A. Forest Serv. Res. Pap. PNW-69, 46 pp., illus. Pacific Northwest Forest and Range Experiment Station, Portland, Oregon.

The disease caused by *Elytroderma deformans* intermittently results in severe local damage. Based on field studies since 1949, disease symptoms, vegetative spread within the host, behavior in sapling stands, and damage to old growth are discussed. Guidelines are given to minimize damage in young stands and to manage mature stands. Despite its alarming appearance, the disease does not threaten the future of ponderosa pine.

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Headquarters for the PACIFIC NORTHWEST FOREST AND RANGE EXPERIMENT STATION is in Portland, Oregon. The area of research encompasses Alaska, Washington, and Oregon, with some projects including California, the Western States, or the Nation. Project headquarters are at:

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